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CONTENTS

American Needs the Older Handicapped Worker. *R. T McIntire* 203
The Challenge of Aging. *R. Harris* 210
Cortisone in the Immediate Therapy of Apoplectic Stroke. *H. I. Russek, B. L. Zohman and A. S. Russek* 216
Adams-Stokes Disease in the Geriatric Patient. *R. F. Sheely* 223
Gout. A Summary of Recent Developments in Therapy. *E. F. Rosenberg* .. 229
Cutaneous New Growths of Geriatric Interest. *F. E. Senear and F. J. Szymanski* 240
Submucosal Lipoma of the Colon. Case Report. *E. P. Russell, J. F. Sulzbach and F. D. Winter* 247
Supervoltage X-Ray Treatment of Carcinoma of the Bladder. *H. F. Hare, J. G. Trump, R. C. Granke and J. Anson* 251
American Geriatrics Society
Program of the 1954 Annual Meeting 256
International Association of Gerontology
Provisional Program of Third Congress 260
Report of Research Committee on Biology and Medicine 262
Medical Library Association Annual Meeting 263

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JOURNAL OF

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VOLUME II *April, 1954* NUMBER 4

AMERICA NEEDS THE OLDER HANDICAPPED WORKER

VICE ADMIRAL ROSS T McINTIRE, (MC) USN, RET.

Chairman, The President's Committee on Employment of the Physically Handicapped, Washington 25, D. C.

Some nineteen centuries ago Galen said that employment is nature's best physician. This is even more true today, particularly as it pertains to the older worker who somewhere along life's bumpy road acquired a disability. Employment of the man or woman with a disability is vital at the ages of 20, 30 or 40. It is just as important at the ages of 50, 60 or 70, assuming that his employment is fitted to the abilities, skills and tolerances of the individual.

The time has long passed when each man or woman worked for himself or herself alone. In our age of crisis the work of individual men and women is closely interrelated and seriously effects our ability as a Nation to survive. There are relatively few jobs today which do not have a cumulative effect upon national security and towards increasing the odds in favor of a world peace with honor and dignity. Even those jobs which may have no direct or indirect bearing upon national needs become critical if they can be filled by people not now in the labor market, thus releasing more people for jobs with a national purpose.

Sometimes the placement of a handicapped person in a job starts a chain reaction that is almost unbelieveable. This is particularly true of an impaired worker in an older age group who has been relegated to life's scrap heap and whose once saleable skills are rotting away from disuse. The added worker increases production of goods, or adds to services in his community. He ceases to draw money from public or private sources, thus releasing this money for other purposes. He begins to pay taxes, thus making additional funds available for government. He once more becomes a buyer, purchasing things previously denied him. He may or he may not release another trained worker for the military service or a higher skilled job vital to defense. He may or may not release one or more people at home from ministering to his needs, thus making it possible for them to make more constructive contributions in the home or in the work force.

In addition to all of these links in the chain, the worker himself finds new purpose in life, greater meaning in himself and his community and a resulting increase in the possibility that he or she will live longer with less drain upon

community welfare and medical facilities now taxed to the breaking point in too many of our Nation's counties.

Since 1947 The President's Committee on Employment of the Physically Handicapped has been engaged in a gigantic informational, educational and promotional task. The results achieved have proved without question that, given the facts, the public will react properly, if somewhat slowly. We have stressed through every possible means the truth of our basic thesis, that "It's Good Business to Hire the Handicapped." In doing this, we have stayed within the Congressional scope of our authority and generally confined our emphasis to the physically handicapped. There are other groups concerned with the mentally retarded and also with those who have advanced in age to a point where the labor market seems unwilling to absorb them.

To the extent that advancing age brings with it many illnesses and disabilities, we have included the older worker in our efforts. This year, in a Foreword to the Program Guide for use by Governors' Committees, community groups and individual citizens, I said:

"The President's Committee is engaged in an educational program that will develop in the minds of employers a rationale for the employment of physically handicapped people, including older workers with disabilities. We must be successful, for, without an established practice of including in the labor force millions of handicapped people who can be made employable, greater economic burdens will be placed upon taxpayers.

"The aging process, which brings about many hidden physical handicaps, is now being given serious study by intelligent medical planning groups. It is to be hoped that information from these studies will be properly applied in conserving manpower so that older handicapped workers can also take their place."

There are those in the employment and rehabilitation field who shy away from too close a connection between the older worker and the handicapped worker. These people feel that an older worker has enough strikes on him already without having to bear the additional burden of having employers thinking of him in terms of also being handicapped. Naturally, a great many people in the older age groups have visible or non-visible disabilities. However, many do not.

As I progressed in years through first one and then another retirement from meaningful service, I have revised my own definition of an older worker. We have all seen workers young and strong of mind and body at 65 and we have all seen those old of mind and broken of body at 45. Hence, chronologic age can no longer be used as a final yardstick in the aging process as it pertains to employment. For myself, my definition of an older worker is "One who is ten years older than I." I dare say that many of our most vital people in their thirties and forties would also accept this definition, along with the sliding scale that goes with it as they themselves advance in years.

Regarding the word "handicapped" as it applies to workers, we have succeeded in large part in changing the definition from that of relegation of the person concerned to a quiet place in the sun out of people's way, to today's definition which claims that there is no job that some handicapped person cannot perform. We must do the same thing with the older worker.

As doctors and scientists increase man's life span, employers and public officials must keep step with them, so that the added years can be made useful, productive and pleasant. Great strides have been and are being made in this direction. More and more firms are thinking today in terms of increasing their use of both older and handicapped workers, always with proper safeguards for them and their fellow workers, the firm and the community.

Several years ago the Chamber of Commerce of the United States and the National Association of Manufacturers sent out questionnaires to a large number of employers in an effort to ascertain attitudes of top management toward the handicapped and the older worker. These surveys resulted in the issuance by both groups of findings, which indicated that there was a proper climate for employment of these two types of employees. Nothing in the intervening half decade has disproved the results of these findings. On the contrary, a similar survey today would undoubtedly reveal greater and greater use and understanding of the capabilities of the older and of the handicapped worker.

The National Association of Manufacturers, an organization not generally associated by the public as working in the fields of welfare and charity, has as one of its cardinal principles in its little booklet, "NAM Believes," the proper employment of qualified handicapped and older workers. The fact that too many businessmen still haven't learned to their own advantage that it's good business to hire the handicapped is no more an indictment of NAM than the fact that too many nominal Christians fail to practice Christianity in the market place is an indictment of the Churches.

The fact that more and more groups and individuals are showing increased interest in the physically handicapped worker is a measure of our own coming of age as a Nation. An irrational devotion to physical perfection did not save Sparta from eventual destruction. We cannot expect to match the hordes of the Kremlin in numbers alone, and we certainly shall not live up to our other possibilities if we continue to lose the productive abilities of millions of potential workers yearly in spite of great progress and unassailable statistics.

We need bigger and better weapons to insure survival, certainly. We need strong air, sea and land forces, certainly. We need great laboratories and great expense in scientific research, certainly. We also must one day realize that we can no longer afford *not* to spend a half billion or a billion on rehabilitation of men, women and children. If we were to spend the average amount it costs to rehabilitate a person into employment, $500, on the 2 million men and women that the Task Force on the Handicapped estimated could be added to our labor force, it would cost us a billion dollars. The amount these people would pay in taxes alone would pay for the one-shot cost of rehabilitation in less than a decade. The amount they would contribute in goods and services, in purchasing power and in taxes saved is incalculable. Suffice it to say that it is so great that we must eventually face up to the question of whether we can afford *not* to spend a billion on rehabilitation of the handicapped. And this question is just as applicable in peace or war or in an in-between period such as today.

There is so much being written today about the employment and rehabilitation of handicapped workers that it is a wonder busy people have had time to as-

similate all the facts and understand all the findings. The same thing might well be said of the aged, including the aged worker with a disability. The Veterans Administration published a 30-page bibliography on the subject "Care of the Aged" for the 1940–1950 period. "Care" is admittedly only one small facet of the subject; yet in order to cover the decade in which we live many more than thirty pages will doubtless be required to record the bibliography.

It is no secret, however, that where a trial has been made, physical medicine and vocational rehabilitation have emptied "chronic" wards of as many as half of their patients within a few months. These people, who were motivated to leave their beds and forget their disabilities and their advancing years, are today living more normal lives at home and working as members of a free society, all because someone fanned the spark that had not yet burned out. It wasn't easy. Nothing significant or important comes without struggle and failures, but these men from Veterans Administration hospital wards are prime test cases, the advance guard if you will, of the 2 million we could reclaim and reuse.

In addition to the volume of material on the handicapped worker, large enough so that the Bibliography of The President's Committee includes a list of several other bibliographies, there is an increasing volume of material on the older worker. Much of the material on employment of the older worker is also significant in our work for the handicapped. Two reports in particular are outstanding:

"Workers Are Young Longer," published by the Bureau of Employment Security, U. S. Department of Labor, and "Employment and Economic Status of Older Men and Women," Bulletin No. 1092 of the Bureau of Labor Statistics, dated May, 1952.

In the former, which is a report of findings on the older worker from studies in five localities, the following quotation is significant because it applies as well to the handicapped as to the aged about whom it was written:

"During World War II, not only did older persons normally in the labor market seek, and obtain employment, but some 750,000 persons who could have retired under the social security old-age and survivors insurance program stayed on their jobs, and some 70,000 persons who had already retired under the program dropped their benefits and returned to work. Moreover, after the war, these older workers did not follow other emergency workers out of the labor market. So far as possible, they remained on their jobs, and continued in gainful employment. As of January 1, 1952, 1,072,000 persons, or about one out of every three persons 65 years and over and eligible for retirement under OASI, remained at work. . . . In wartime, the stimulus of patriotism might have kept older workers in the labor force, but their staying at work after the end of war proved that some other factor dominated their activity. Clearly, many older workers prefer employment even when entitled to retire. . . ."

With reference to the prevalence of disability among older workers, the employment service people had this to say:

"The studies involved a comparative analysis of the characteristics of older workers among the registered unemployed. This analysis indicated, among other things, that the incidence of physical disabilities which are real vocational handicaps among older workers is no greater than among younger groups."

TABLE VI*

Proportion of Physically Handicapped, by Age Groups, in Active Files of Three Study Localities in Typical Month of 1950

Age Groups	Percentages		
	Columbus	Lancaster	New York
All ages	9	3	4
Under 45	6	3	4
45 and over	4	3	3

TABLE VII*

Types of Handicaps of Applicants Over 45 Years Ranked in Order of Frequency

	Rank		
	Columbus	Houston	New York
Vision, Hearing, Speech	1	1	1
Trunk, Spinal, Abdominal	2	2	3
Cardio-vascular	3	5	2
Upper Extremities	4	4	4
Lower Extremities	6	3	5
Neuro-psychiatric	5	7	7
Generalized or Systemic	7	6	6

* Reproduced with permission from "Employment and Economic Status of Older Men and Women", Bulletin No. 1092, Bureau of Labor Statistics. Washington, D. C., Govt. Printing Office, 1952.

TABLE VIII*

Proportion of Handicapped in Samples as Compared to Proportion of Handicapped Who Obtained Employment

	Percentages			
	Columbus	Houston	Lancaster	New York
Handicapped in sample	15	29	1	13
Handicapped employed	16	22	.2	17

The report continues, "Our studies showed that . . . the presence of handicaps in older workers does not in itself decrease the likelihood of their finding new jobs. Evidence of this is that the proportion of handicapped older workers in the study groups who obtained employment was everywhere very close to the proportion of handicapped in the study samples, and in Columbus and New York was greater than the proportion in the samples. (Table VII)

"Probably the handicapped older worker has, in most cases, adjusted to his disability, so that it is no longer a significant factor in his competition for employment. This is possibly because the most prevalent handicaps among older workers are of types which result from very gradual deterioration rather than from sudden accident or acute illness. . . .

"Recent studies of the medical and psychological aspects of aging bear out the conclusion of the employment service studies that older workers are not necessarily less physically able than younger workers. . . ."

Although the Labor Statistics Bulletin contains fifty-eight pages of charts, graphs and statistics, one remark by Commissioner Ewan Clague points up the continued problem of the older worker. He said:

"There are serious gaps in available information on significant aspects of the problem of older workers. The lack of this information emphasizes the importance of still further research needed to clarify existing knowledge."

It has been in this vital area of clarifying existing knowledge as well as in carrying out further research that The President's Committee has been most useful to the Nation, employers and employees alike. Through working relationships with Governors' Committees on employment of the physically handicapped in all States and Territories, The President's Committee has not only been able to lead and stimulate, but to compile and distribute the results of research in the States and communities. This research is not the pure research of the laboratory, but the popular type found in company reports, statements of executives, medical department operating manuals and plant procedures for counseling, testing and selective placement of the handicapped. It is also in the area of group dynamics, the function and success or failure of interested and coordinated groups at local, State and national levels of society, that results of great import have been discovered and analyzed.

The aging process provides not only opportunity but a responsibility for the men of medicine to provide in a measure the answer to increased longevity. Preventive medicine finds a fertile field in averting the crippling symptoms of cardiovascular disease. Neurologic diseases plague the older worker. Other hidden handicaps make employment difficult. Epilepsy and that great crippler, arthritis, add to the list.

From the standpoint of economy it is necessary that a better approach be made to the employment of people who are growing old. The national economy is directly affected by increased longevity. Old-age pensions are constantly increasing. There are today over 13½ millions of people over the age of 65. Less than 1½ million are employed in any fashion. Of course, many are too badly handicapped physically for employment, as employers now understand the term. Physical medicine, rehabilitation and proper selective placement can do much to add hundreds of thousands to our national manpower potential.

It is estimated that no more than 15 million men are available for military service. Industrial needs and failure to meet physical standards are the principal reasons. What then of the physically handicapped? They must be included in the employed force of the Nation. At least 2½ million are available for rehabilitation and training. This would give a backlog against a manpower shortage in time of national emergency.

The economy cry is heard when this subject is presented.

The high cost of rehabilitation? Yes, it does cost more money. For example—and these are the very latest figures, obtained in the last week—the cost of operat-

ing the State Federal program of vocational rehabilitation during 1953 was $564 per person rehabilitated.

Admittedly it costs money to rehabilitate an older person, but the returns on the investment are considerable. Most rehabilitated younger persons had some sort of income before learning skills under the rehabilitation program. Most older persons, on the other hand, had virtually no gainful activity prior to rehabilitation, so that their new income became almost 100 per cent new productivity with its attendant benefits to the economy and society as a whole.

Now let us turn to a factual report from Buffalo, New York. Of 5,100 persons rehabilitated and employed, the study showed the average monthly income to be $183 before injury. Following rehabilitation and employment in the new skill the average monthly income was raised 71 per cent or to $318.50. Good business for local communities is reflected in the increased buying power of the individual.

The Buffalo story is an example of what can be done for the handicapped older worker, whether the handicap is caused by disease or by injury.

As we consider our problem of employing the aging citizen let us apply the following principles:

1. We aid conservation of our human resources.

2. We raise the dignity of the individual by making him a useful citizen of his community.

3. We strengthen the self-respect of the older citizen by proving he is still useful.

4. We make use of the experience and wisdom that can be so helpful to the youth.

5. We prove that it is good business to employ qualified handicapped people. All of this falls into the pattern that is typically American—helping people to help themselves.

It is obvious that among our 158 million citizens there is a hidden treasure of manpower among our aged. Proper use of this hoard can, in the next few years of vital emergency, place our economy on a sounder basis than ever before. Any method of filling the industrial manpower demand and thus releasing other men for potential service to their country is sound, practical business as well as good patriotism.

The older worker, within the limits of his capacities and skills, can prove himself if given a chance. This has been demonstrated in thousands of instances. There are still more thousands of such persons who should be given an opportunity. Their lives will be made richer, and their declining years more dignified and happy by such a move.

The employers of this country who have vision will not fail to take the foregoing facts into consideration. Any formula that helps the individual, helps the community, and at the same time does not violate in any way the basic principles of good business—to make money—should have a strong appeal to the industrial leaders of this country.

THE CHALLENGE OF AGING*

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Today the challenge of aging is one of our most perplexing problems. Although no fountain of eternal youth has been discovered, medical and public health advances during the past half century have increased our life expectancy at birth from forty-nine years in 1901 to sixty-eight years in 1949. Since 1900 the population of the United States has doubled while the number of persons 65 and over has quadrupled. There are now 13 million men and women 65 years and older in the United States (1). This group, increasing currently at the rate of about 400,000 a year, has new medical, social and economic needs which challenge our complex industrial civilization. Concepts and policies satisfactory for a young pioneering nation require dynamic revisions to meet the growing predicament of an older population.

Lake Placid is a symbolically significant site at which to attack this problem, for its precambrian rocks, over 1100 million years old, are also aging. Compared with the geologic age of this majestic and thrilling setting, the lifespan of man is an infinitesimal speck on the eternal calendar of time. Yet we are more concerned with the aging of man, since it represents a realistic challenge that must be met cooperatively and imaginatively if our brilliant medical, social and economic evolution is to progress.

The crux of the aging problem is the period of retirement, at which time changing social roles and artificial patterns of activity force us to withdraw from the occupations or activities which have contributed the very reason for our existence (2). This is the "Fixed Period" of Anthony Trollope and Sir William Osler when the mature individual must leave the roller coaster of life, usually without having made adequate preparation for retirement. Frequently this person finds himself with time on his hands. If he cannot meet the challenge of retirement, he becomes a burden to himself, his family and his community. This fixed period of retirement is characterized by other serious problems, including the maintenance of health, a reduced income, the loss of human companionship, and often the need for a change in living arrangements since, as Shakespeare noted "Crabbed age and youth cannot live together."

The ubiquitous challenge of chronic disease is the most urgent medical problem of aging, because of its increasing incidence and economic consequences. A chronic disease is an impairment in health that requires an extended period of medical supervision with ambulatory, home, hospital or other institutional care.

* Presented at the 49th Annual Health Conference, New York State Department of Health, Lake Placid, N. Y., June 1953.

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Disability results from an impairment of the biologic, physiologic or social efficiency of the individual, preventing him from pursuing his usual activities (3).

The mounting toll of chronic disease is nature's price for man's increased longevity. Chronic disease causes more than 2 out of every 3 deaths in the United States. In New York State the number of deaths from such conditions as diabetes, cancer and heart disease has jumped from 25 per cent of all deaths in 1900 to 75 per cent of all deaths in 1950 (4). Chronic disease weighs especially heavily upon older people who suffer from various types of heart disease, arteriosclerosis, nervous and mental disorders, arthritis and tuberculosis. As a result, these persons require more medical care, often of a highly specialized and costly type.

The economic consequences of chronic disease are important, since older people constitute a group in which large numbers have little or no income. Although 15 per cent of families headed by persons 65 years of age or over have annual incomes in excess of $5,000, the median annual income of such families is $1,903. For those living alone or with non-relatives it is $646 a year (1).

Between the Scylla of inflation and the Charybdis of reduced income the senior citizen is in the unenviable position of trying to pay increasing medical and hospital expenses without sufficient funds. Although old age assistance and social security payments may help, they are only drops in the sea of medical expenses. Frequently our senior citizens must seek relief from welfare agencies in order to meet their financial burdens.

The overwhelming problem of medical and hospital expenses for older people must be faced. One solution may be voluntary hospital insurance plans for the elderly; another may be free hospitalization for persons over 65 years of age. Another and better answer is for the senior citizen to work as long as his health and ability permit. In many instances this would mean employment up to and even beyond the age of 70. Let physiologic rather than chronologic age be the yardstick for retirement.

Still another approach to the problem of medical care for the aging citizen is the reduction of costs by the greater use of home-care programs and properly managed nursing homes, by the construction of hospitals for the chronically ill, and by the modernization of old-age homes. Elderly patients with chronic diseases need not be maintained in expensive hospitals geared primarily for the care of the acutely ill patient. They will get along far better at home or in institutions designed for them, institutions in which they can be rehabilitated first physically and mentally, and then economically and socially. They should not be isolated, but rather integrated into the life of their community, with benefit to both. The establishment of such facilities means an initial outlay of funds. We all know, however, that "one must spend money to make money." Similarly one must spend money to save money.

At the Ann Lee Home we have demonstrated the usefulness of such a program. We have inaugurated a policy of rehabilitation which starts with complete

medical examination and treatment and then attempts to restore the senior citizen to usefulness by physical and social integration. In many patients we have been successful. We have projected our rehabilitative efforts into the field of social relations by utilizing the Albany Senior Citizens Center, a day center for older persons. Our county home residents visit the Center in Albany frequently and participate in its program. Although they are reluctant to go at first, later they are enthusiastic and desire to return voluntarily. In the future we hope to extend our efforts into the vocational field with the help of developing community resources, such as a sheltered workshop. In this way we can help the older person to help himself to better health and wealth.

Aging challenges the interest and imagination of the physician, the nurse, the nutritionist and the research worker. For the practicing doctor it creates a new field of medicine—geriatrics, or the medical care of the aging person. With the increasing number of older people the physician may expect to treat more elderly patients. He must recognize that their medical problems may differ from those of younger people. Illnesses from which the younger person easily recovers may be grave for the older person, because the latter has a poorer reaction to stress. Greater use of diagnostic equipment is necessary in geriatrics, because the clinical signs and symptoms of illness are often diminished, and may mislead the physician. At the Ann Lee Home we have been able to detect many previously unsuspected medical conditions with the routine use of adequate laboratory facilities. For example, we uncovered numerous cases of active pulmonary tuberculosis among our residents when we began making routine x-ray examinations of the chest. Similarly we found a large number of anemias, following the inauguration of routine blood counts. As a result of these thorough diagnostic studies performed routinely, we have been able to restore many of our residents to better health than they have enjoyed for years. The doctor treating geriatric patients should be aware of the value of general supportive measures, as well as of specific therapy. He must recognize the importance of preventive medicine and proper nutrition throughout life and be cognizant of the social and economic forces which influence the health of his aging patient. He should be willing to utilize the important ancillary services of the nurse, the nutritionist and the social worker. As a member of this health team the physician will find treatment of the older person satisfying and regarding.

The well trained nurse in geriatrics not only follows the specific orders of the doctor, but also performs the implicit duties which are equally important. By a few cheerful words or an encouraging smile she can strengthen the sagging morale of the bedridden invalid, giving him hope and happiness. The nurse is also important in any treatment program of the elderly patient, for she can cajole him into taking sufficient nourishment and can encourage him in his rehabilitative efforts.

The nutritionist also experiences the challenge of aging. As assistant medical director and cardiovascular consultant at the Ann Lee Home in Albany, I have been deeply appalled by the poor state of nutrition in the patients admitted to our county institution, regardless of whether they have been picked up in the

streets, moved from another nursing home, or transferred from a hospital. About 70 per cent of our patients have signs and symptoms of malnourishment and avitaminosis on admission. Frequently they are semicomatose because of dehydration, anemia and electrolyte imbalance. When fluids, vitamins and other drugs are administered to correct their nutritional deficiencies, most of them respond readily.

Why is this state of malnutrition so common in our aging population? It exists, not because our present knowledge of nutrition is inadequate, but because the older citizen frequently has neither the money nor the knowledge necessary for proper nourishment. Since the maintenance of health is cheaper than the treatment of disease, preventive geriatrics should emphasize proper nutrition. In this field it is certainly true that an ounce of prevention is worth a pound of cure. Health conservation throughout maturity means less gambling with health in old age. The health of older men and women can be better conserved if we emphasize the need for better nutrition at all ages.

In general, geriatric patients are malnourished for five main reasons, namely, primary dietary inadequacy, poor absorption of food, diminished utilization of absorbed foodstuffs, increased nutritive requirements, and increased excretion of foodstuffs.

Primary dietary inadequacy may result from poor food habits, excessive consumption of highly refined foods or actual poverty. Poor food habits may either be present throughout life or develop with the onset of old age. Lack of proper nutritional education may have led to these habits. Frequently older people undergo a decline of their sense of taste, which reduces the appetite and the incentive for eating a great variety of foods. Because they lack sufficient teeth, they often become food faddists, always eating the same foods which do not require chewing. Many of these people eat too little meat and consume excessive amounts of highly refined foods, especially carbohydrates. Rich sources of vitamins are frequently neglected, in favor of food which increases the requirements of vitamin-B complex. When actual poverty exists, aging persons eat less meat and other protein foods. They fail to ingest sufficient protein to supply the essential amino acids which keep their body tissues in good repair. They may have a lower resistance to disease and become more apathetic and less ambitious. Should surgical treatment be necessary, such protein-depleted patients are poor operative risks.

Poor absorption from the gastrointestinal tract is also responsible for the malnourishment of many elderly people. Although it has been said that we are what we eat, it is more accurate to say that we are what we digest. Elderly people may digest food poorly because of poor chewing habits and the decreased secretion of digestive juices which accompanies aging. Poorly digested foods are not well absorbed. Changes in the absorptive capacity and motor function of the intestinal tract may further hinder proper absorption in old people.

Geriatric patients with chronic disorders such as arteriosclerotic heart disease, liver dysfunction and diseases of the gastrointestinal tract are also likely to show signs of nutritional deficiencies as a result of either inadequate absorption or

inadequate utilization of vitamins. Furthermore, some conditions, such as fever, may increase the nutritive requirements or result in the excretion of essential nutrients.

All these factors lead to malnutrition in aging patients. We can improve this state of affairs by emphasizing the need for better nutrition at all ages. We can help our older patients to select low-cost foods which will provide satisfactory nourishment. We can correct their medical conditions so that they may utilize their foods properly. Malnutrition in geriatric patients must be overcome if we are to lower the incidence of chronic disease and improve the health status of the senior citizen.

To the research worker, aging offers a twofold challenge. The first is to study the causes and treatment of chronic illness; the second is to determine what aging actually is. Scientists all over the world are working on the first challenge. Work on the second challenge is more limited, but is beginning to gain impetus. Research must set up new standards for aging, since too many of the alterations commonly attributed to senility, such as skin changes, weakness or debility, have proved to be nothing more than nutritional deficiencies. Chronic disease also may cause some of the changes we call aging. It is interesting to learn that bacterial organisms normally inhabiting the gastrointestinal tract of rats may induce liver changes associated previously with aging. Research workers at Notre Dame have found that in germ-free rats maintained in a germ-free environment for generations these liver changes do not develop with aging, unless bacteria are introduced into the gastrointestinal tract (5). Such studies show the importance of establishing new yardsticks by which we may measure the aging process. The research worker has untapped reservoirs of human material in our old age homes, our hospitals, and our research institutions.

CONCLUSION

Aging has become a major individual and national problem because of the impact of our increasing longevity upon an industrial civilization with fixed concepts and policies. The overall challenge is the creation of a climate in which there can be a new phase of growth and development for aging citizens, so that these individuals with their broad experience can render new contributions to our dynamic culture in the leisure of their retirement. This challenge requires the expansion of local medical services, nursing homes and proper hospital facilities; the establishment of adult educational and guidance courses which help older people to make intelligent and sensible adjustments to the realities of old age; the establishment of community centers for recreation, social contacts and creative development of senior citizens; job-counseling services to aid older workers; and the formation of health teams, composed of doctors, nurses, social workers, nutritionists and community leaders, to develop effective community action for the solution of these needs of older people. All these services will help to create the sunny climate in which our aging population can take firm root and thrive.

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CORTISONE IN THE IMMEDIATE THERAPY OF APOPLECTIC STROKE

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In recent years considerable progress has been made in the rehabilitation of the hemiplegic patient. The brilliant work of Rusk and associates (1) and others has clearly demonstrated that modern methods of rehabilitation and retraining can successfully restore such patients to lives of independence and productivity. The advances in the therapy of residual defects, however, have far exceeded the questionable gains in the treatment of the acute phase of the cerebrovascular accident.

The immediate treatment of "stroke" is mainly devoted to measures for saving life and restoring physiologic balance. All authorities stress the importance of skillful nursing care, maintenance of hydration, attention to bladder and bowels and the prevention of hypostatic pneumonia. The current attitude toward therapy has been summarized by Alvarez (2) in a recent editorial as follows:

"The best treatment for cerebral thrombosis is an expectant one. We all know that in many of these cases a weakness of the arm or leg clears up, and often aphasia disappears almost entirely. This recovery may be due to subsidence of edema of the brain or it may be due to rerouting of impulses around the destroyed area of the brain tissue."

Nevertheless, despite the recognition that return of function often occurs spontaneously due to a clearing of cerebral edema and improvement in cerebral blood flow, there is impatience with the necessity for awaiting "expectantly" the final and unalterable decree of nature. The hope of decreasing the degree of neurologic residua has stimulated interest in a more active treatment of cerebrovascular occlusions. Thus, attempts have been made to improve the cerebral circulation in such patients by the administration of vasodilator drugs, by the injection of procaine into the stellate ganglion, and by the administration of anticoagulants. Drugs of the vasodilator group have been found of little value for treatment of organic occlusion of the cerebral vessels: on theoretical grounds alone, there is little to suggest that any lasting benefit could result from a short period of vasodilation. When neurologic signs and symptoms are the result of transient cerebral vasospasm, however, large doses of papaverine (3 to 8 grains, three or four times a day) do have great value in the prevention of recurrences (3).

There is a divergence of opinion in the literature as to the efficacy of blocking the stellate ganglion. The difficulty in differentiating between clinical improvement resulting from therapy and that occurring spontaneously, indicated the need for carefully controlled studies. It has been argued that no logical basis exists for blockage of the stellate ganglion, since most observations on the circu-

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lation of the brain show that the cerebral vessels are not under the control of the cervical sympathetic nerves. Scheinberg (4), and Harmel and associates (5) actually found no alteration in cerebral hemodynamics following blockage of the stellate ganglion, but it has not been established whether the patients in their study were comparable to those who receive such therapy for clinical purposes.

Anticoagulants have been tried in patients with cerebral thrombosis in an effort to prevent further extension of pre-existing thrombi or the formation of new clots. In cases of hemorrhage or certain blood dyscrasias, the danger of this form of therapy is obvious. It is also believed that the risk of secondary hemorrhage in a softened area of the brain is increased if the clotting mechanism is disturbed (6). Consequently, no definite conclusions are warranted at present regarding the value of this form of treatment.

From these considerations, it would appear that no acceptable therapy for "stroke" is now available to hasten recovery from the acute phase of the attack, or to reduce the extent of the cerebral lesion and its associated neurologic defects. In previous studies (7) concerned with the rehabilitation of hemiplegic patients, the authors observed that the administration of cortisone promoted a sense of well-being, improved motivation, stimulated psychomotor activity and diminished pain. These desirable actions of the drug permitted early ambulation and shoulder manipulation even in neglected cases with painful contractures. In all of these patients, cortisone was administered many weeks or months after the development of the acute cerebrovascular occlusion. The possibility that cortisone may also have value in the acute phase of the disease immediately following the onset of "stroke" was suggested, not only by these findings but by other considerations of a theoretical nature. Many workers (8) have demonstrated that following administration of cortisone, there is delay in fibrosis, diminished cellular reaction, scanty formation of ground substance, little capillary formation and complete lack of fibrin and exudate. Inasmuch as local cerebral edema surrounding an area of infarction is commonly regarded as a contributory factor in the perpetuation of neurologic signs, the retardation of the mesenchymal tissue response would appear to be a most important and immediate objective of therapy. If this retardation could be achieved, nerve cells which otherwise would eventually die of anoxia as a result of the tissue reaction in the zone surrounding the affected area, might be spared and the ultimate irreversible changes in the brain might be held in abeyance or reduced to a minimum. The probability of such an effect from cortisone in cerebral infarction is strengthened by observations of its influence on tissue response to experimentally produced myocardial infarction in animals (9).

It appeared, therefore, that cortisone might possess an unusual combination of desirable effects for the patient sustaining an acute cerebrovascular accident. Such benefits, however, would have to outweigh the risk of undesirable side effects which might be encountered in the use of this drug. Cosgriff (10) and Steinbrocker and associates (11) have warned of the danger of vascular complications among patients receiving cortisone, but our own experience with prescribing the drug for many patients with obvious thrombotic propensities has failed to

confirm this risk. Hypertension and diabetes mellitus also presented no insurmountable difficulties, when simple precautions were taken. Possible untoward effects from a relatively short course of cortisone, therefore, did not seem of sufficient importance to preclude its use in patients sustaining an acute cerebrovascular accident. The present paper is concerned with the results of such treatment in 15 consecutive cases of this disorder.

CLINICAL MATERIAL AND METHODS

Description of Patients: Of the 15 patients in this series, 12 were male subjects and 3 were female. The ages ranged from 42 to 74 years, with a mean age of 60.8 years. Hypertension was present in 11 of the 15 patients. A history of angina pectoris was obtained in 5 and previous myocardial infarction in 4. The cause of the cerebrovascular occlusion was diagnosed as thrombosis in 9 cases, embolism in 3 and hemorrhage in 3. Lumbar puncture revealed blood in the spinal fluid in the 3 cases with hemorrhage. Embolism occurred in 2 patients convalescing from acute myocardial infarction and in 1 patient with rheumatic heart disease and chronic auricular fibrillation. Six of the total group had previously suffered from bouts of congestive heart failure. Diabetes mellitus of mild to moderate severity was present in 3 of the cases. Three patients (all with cerebral hemorrhage) were in coma when treatment was instituted. In 6 additional cases the signs and symptoms were also severe; in only 3 instances were they mild. Six patients showed marked right hemiparesis, conjugate deviation of the eyes and varying degrees of clouded consciousness. Marked facial weakness, aphasia or dysarthria and difficulty in swallowing were found in several of the latter. Three patients in the series showed a mild left hemiparesis.

Administration of medication: In most instances 300 mg. of cortisone was administered orally in divided doses on each of the first two days, with progressive diminution to a maintenance dose of 50 mg. daily through the third week. In 3 patients the course of cortisone was intentionally discontinued at the end of the first week but in 2 of these it was resumed after several days. Cortisone was administered parenterally in patients who were comatose or who had difficulty in swallowing. In addition, when possible, each patient was given a low-salt diet with 3 grams of potassium chloride orally a day in divided doses. Serial observations were made of the electrocardiogram, hemogram and urinalysis. Repeated daily determinations were made of the blood pressure and heart rate. Serum sodium and potassium levels were not followed. In all patients cortisone was started within forty-eight hours after the onset of the cerebrovascular accident; in 6 of the 15 patients it was begun within twenty-four hours after the appearance of symptoms. In 3 patients (2 with cerebral embolism and 1 with cerebral thrombosis) cortisone was administered after blockage of the stellate ganglion proved ineffective.

RESULTS

In cerebral hemorrhage: In the 3 cases in this group cortisone appeared to have no demonstrable beneficial effect. All 3 patients were moribund when treatment was instituted. Coma deepened and death occurred within two to five days.

In cerebral thrombosis and embolism: In 9 of the 12 patients in this group dramatic clinical improvement was noted within twenty-four hours after the commencement of cortisone therapy. There was not only a striking amelioration of paralytic signs and symptoms but also unquestionable benefit from changes in the mental, emotional, sensory and psychomotor status. Conjugate deviation of the eyes, incoherent speech, dysarthria and aphasia appeared to clear rapidly during cortisone therapy. The greatest effects were noted among those who were somnolent, stuporous, mentally depressed or apathetic. In 9 patients so afflicted, these symptoms were replaced overnight by a keen alertness and sustained interest in personal progress. Accompanying these mental changes there was noteworthy subsidence in the degree of paresis of affected extremities as shown by significant increase in the range of motion and in the ability to perform various activities. Moreover, active rehabilitative measures were easily instituted after only one or two days of treatment, as a consequence of the rapid recovery from acute mental disturbances, associated with improved motivation, psychomotor stimulation and increased sense of well-being. The greatest clinical improvement in all cases occurred within the first twenty-four to forty-eight hours after cortisone was started; thereafter, definite but more gradual return of function ensued. The most marked benefits from the drug were observed in the more severe cases of cerebral thrombosis and embolism. Cortisone was intentionally discontinued in 3 cases after approximately one week of therapy. In 2 of these, paresis increased in the involved extremities after the drug had been withheld for two and three days, respectively. The readministration of cortisone, however, was soon followed by improvement in the motor power of the affected limbs. No such rebound phenomenon was observed at the termination of therapy in any of the patients receiving cortisone continuously for the three-week course. Of the 12 patients with thrombosis or embolism, 4 were walking unaided or with a cane after three days of treatment, 4 more were ambulant at the end of a week and 2, after ten days of therapy. The remaining 2 patients were intentionally confined to bed because of the coexistence of acute myocardial infarction, but even in these the psychologic state of the patient and the improvement of function in the involved lower extremity would have permitted ambulation at a relatively early date. Remarkable neurologic recovery had occurred in all patients in this group by the time the three-week course of cortisone was completed. Residua consisted of mild to moderate weakness in the hand-grip, lower facial paresis and minor difficulties in speech and deglutition. Only 1 patient showed continuing severe monoplegia of the upper extremity. No serious difficulties were encountered from the use of cortisone in this study.

COMMENT

It is acknowledged that a rapid and nearly complete recovery may occur after a cerebrovascular accident with nothing more than symptomatic treatment. Nevertheless, the frequency of prompt and striking improvement in neurologic symptoms in our patients with cerebral thrombosis or embolism during cortisone therapy must be regarded as more than coincidence. Cortisone appeared to convert the discouraged, apathetic or bewildered hemiplegic to a cooperative,

hopeful and even mildly euphoric patient over-night. Clouded consciousness, stupor and disinterest in surroundings were often replaced within twenty-four hours by a degree of alertness and insight not encountered at any time in apoplectic patients treated expectantly. The clinical impression obtained was that cortisone accomplished in one day what ordinarily might take several weeks under conservative treatment. These beneficial changes in the psyche of individual patients in response to cortisone seem indisputable and leave little doubt as to the value of this drug in this disorder. Moreover, the marked improvement in paralytic signs and symptoms which frequently accompanied the striking mental changes may also prove to be directly attributable to the action of cortisone. Supporting this view was the observation of increasing paresis in 2 of 3 patients in whom cortisone was prematurely discontinued, with reversal of effect when the drug was resumed.

Under cortisone therapy, rehabilitative measures can be instituted with little difficulty after only one or two days of treatment, because of the patient's alertness of mind and euphoria. The early employment of retraining methods undoubtedly played a part in the remarkable neurologic recovery observed in most of these cases but, since the major response was observed before the application of such measures, a cortisone effect on the apoplectic lesion in the brain itself should be considered. It is of interest that cortisone appeared to exert its maximum influence in the more seriously ill patients with cerebral thrombosis or embolism. Milder cases responded satisfactorily but the most dramatic effects were noted in patients with marked hemiparesis, aphasia, conjugate deviation of the eyes and clouded consciousness. These findings support the impression that the immediate effects of cortisone are more marked in patients with the more extensive lesions of the brain in which local edema plays a prominent part. Obviously, any method capable of preventing or eliminating the interstitial edema which is an outstanding gross and microscopic characteristic of apoplectic lesions, would preserve nerve cells and permit a greater degree of neurologic recovery. The probability of such an effect from cortisone is further suggested by observations of its action on experimentally produced myocardial infarcts in animals (9). Thus, in the treated animal there was a sharp line of demarcation between the area of infarction and the adjacent muscle, whereas in the control there was a broad, irregular zone of healing. Postmortem injection studies furthermore demonstrated a greater development of collateral circulation under the influence of cortisone. Regardless of the explanation, administration of the drug appears to counteract some of the immediate effects of "stroke" due to cerebral thrombosis or embolism. In a disease which is commonly viewed with pessimism and treated at best with "good nursing care," cortisone should prove a most valuable addition to therapy and a useful adjunct for early rehabilitation.

In the present series of cases no significant complications were observed from the administration of cortisone. In patients with diabetes mellitus, the diabetes was easily controlled by meeting the increased demand for insulin. Clinical signs of sodium retention were not observed and hypertension was not aggravated by its use. Although 86 cases of cardiovascular or cerebrovascular disease have

been treated with cortisone to date by the authors (7, 12 13,), no thrombotic incident has occurred in any case during the course of therapy or in the immediate post-treatment period. This experience in patients with obvious thrombotic tendencies and with advanced vascular disease does not confirm the alleged risk of thromboembolic complications from the use of this drug.

SUMMARY AND CONCLUSIONS

Various attempts have been made to improve the cerebral circulation in patients with apoplectic stroke, in the hope of decreasing neurologic residua. The uncertain responses with such methods have left no alternative to "expectant" therapy. Inasmuch as cortisone has been found of value in the rehabilitation of hemiplegic patients, through its action in enhancing motivation and promoting a sense of well-being, it was considered logical to make a trial of the drug for the acute cerebrovascular accident also. Moreover, since local cerebral edema is believed to be a contributory factor in the perpetuation of neurologic signs, and cortisone has been shown to retard the mesenchymal tissue response to various forms of trauma, additional benefit from cortisone seemed possible in this respect. With these objectives in mind, 15 consecutive cases of apoplectic stroke were treated with this drug within forty-eight hours after the onset of symptoms. The following observations were made:

1. Cortisone was without benefit in the 3 patients in the series who had a cerebral hemorrhage.

2. Cortisone produced striking effects in 9 of the 12 patients with hemiplegia due to cerebral thrombosis or embolism. Within twenty-four hours after its administration a dramatic change was noted in the mental and emotional stature of these patients. Apathy, mental confusion or depression, somnolence or stupor gave way to an alertness of mind, exhilaration or mild euphoria. As a result of psychomotor stimulation, improved motivation and sense of well-being, active rehabilitative measures were effectively instituted without difficulty after only one or two days of cortisone therapy. Accompanying these mental changes, there was rapid improvement in paresis of the extremities, aphasia and conjugate deviation of the eyes. Remarkable neurologic recovery had occurred in all patients by the end of the third week of cortisone administration.

3. Cortisone is a valuable addition to the therapy of acute cerebral thrombosis or embolism. The drug appears to offer an active approach to limiting the assault of a disease which until now has been viewed, at best, with expectancy and, at worst, with defeatism.

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ADAMS-STOKES DISEASE IN THE GERIATRIC PATIENT*

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Since Adams (1) in 1827 described his "Cases of Diseases of the Heart Accompanied with Pathological Observations" and Stokes (2) in 1846 reported his "Observations on Some Cases of Permanently Low Pulse," there have been relatively few case reports of Adams-Stokes disease. About the time Stokes' paper appeared and focused attention upon this disease entity, an important discovery was made in physiology, *viz.*, the effect of vagus nerve stimulation on the cardiac rate. For the next fifty years, the syndrome of a slow pulse rate associated with periods of dizziness followed by loss of consciousness and at times convulsive seizures was thought to be due to vagus nerve excitation. It was not until the turn of the twentieth century, with the application of electrocardiography to the study of these cases, that the intrinsic myocardial lesion was discovered.

Nearly a century passed from the time of Stokes' original paper until the electrocardiographic criteria of Adams-Stokes disease were definitely established. It was in 1941 that Parkinson, Papp and Evans (3) analyzed the composite picture of this disease entity and defined the EKG criteria for diagnosis. These investigators distinguished three separate categories of arrhythmias or combination of arrhythmias originating in a heart affected with a partial or complete heart block, producing attacks of Adams-Stokes disease. These arrhythmias may be listed as follows: 1) ventricular standstill or asystole, 2) ventricular standstill preceded by ventricular tachycardia, 3) ventricular tachycardia and/or ventricular fibrillation without ventricular standstill.

Complete heart block was the basic myocardial lesion in the majority of the 64 cases meeting the foregoing requirements, reported in the literature until 1941. Eighty per cent of the cases with complete heart block had Adams-Stokes attacks with the onset of ventricular standstill. Ventricular tachycardia preceded ventricular standstill in 45 per cent of these cases. Although it has been shown that the conduction to the ventricle is made worse by an increase in the auricular rate, the influence of the latter in the production of ventricular standstill is unknown (4).

Complete heart block occurs in a variety of pathologic and therapeutic conditions which affect the junctional tissue (A-V node and bundle of His) of the conduction system of the heart. Complete heart block has been reported due to fibrosis of the junctional tissue secondary to coronary atherosclerosis (5, 6), calcification of the interventricular septum (7), calcific aortic stenosis (8, 9), fatty infiltration (10), primary tumor of heart (11), phlebosclerosis (12), digitalis

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poisoning (13), congenital heart disease (14, 15, 16) or myocardial infarction (17). In a variety of systemic infections (11, 18, 19, 20), complete heart block has been noted; these infections include tuberculosis, rheumatic fever, viral pneumonia, poliomyelitis, scarlet fever, mumps, whooping cough and German measles. Campbell (21) concluded, after study of 64 cases of complete heart block, that 72 per cent of the cases were between 50 to 70 years of age, and Schnur (22) likewise found 80 to 90 per cent of the cases of complete heart block in the atherosclerotic group. It is only logical to assume that with the increasing numbers of persons in the geriatric group, the incidence of complete heart block will increase

The following two cases illustrate some of these points.

CASE REPORTS

Case 1. The patient was an 83-year-old woman admitted to the Medical Service of Annie Warner Hospital on October 2, 1951, because of a recent "little stroke" and frequent "psychomotor seizures" of two weeks' duration. There was no history of rheumatic fever. Physical examination disclosed an obese female, mentally confused and restricted to bed. The blood pressure was 300/70; the pulse was 38 to 56 per minute and very irregular. Arcus senilis was present bilaterally. The thyroid was not enlarged. The chest was emphysematous and crepitant rales were detected bilaterally at the bases. The apex beat of the heart was beyond the midclavicular line on percussion. The liver edge was palpable below the rib margin. No arterial pulsation was palpable in either foot. The arteries at the wrist were hard and stood out plainly. Patellar, Achilles and Babinski reactions were normal.

Laboratory studies were reported as follows: Urinalysis—albumin, 4 plus; specific gravity, 1.023; 15 to 20 hyaline and granular casts and 4 to 5 RBC per LP field. The blood sugar level was 190 mg., calcium 12 mg., and urea nitrogen 43 mg. per 100 ml. The Wassermann reaction was negative. Hemogram—RBC, 4,000,000 per cu. mm.; WBC, 9,300 per cu. mm.; and Hb, 12.5 grams. The electrocardiogram showed a complete heart block with a varying ventricular rate of 38–54; the QRS-interval was .1 sec. and the Q-T interval was .48 sec.; T-waves were diphasic in L_1, $V_{5\text{-}6}$, aV_L and aV_F.

During the first 24-hour period of hospitalization, the patient suffered 17 attacks of unconsciousness lasting from 30 to 240 seconds. These attacks followed a similar pattern of sudden hesitation in breathing which became labored and later stertorous. Unconsciousness ensued, with marked cyanosis of the face, hands and feet. The pulse was undetectable during these attacks. Although breathing gradually improved, along with detectable pulsation, unconsciousness persisted for a period up to four minutes. Until her death, seven days later, she suffered 7 to 10 attacks daily, brought on when she attempted to swallow food or medicine, or rolled from one side of the bed to the other in the attempt to sit up. Ephedrine sulfate, adrenalin, Pronestyl (procaine amide hydrochloride) and intranasal oxygen had very little effect in controlling the attacks.

Autopsy was performed several hours after death. The heart showed marked left ventricular hypertrophy. High in the interventricular septum, in the approximate area of the junctional tissue, were small multiple areas of fibrosis. There were areas of fibrosis in the posterior wall of the left ventricle. The postmortem diagnoses, following histologic study, were: left ventricular hypertrophy, rheumatic mitral valvulitis (inactive), myocardial degeneration, focal fibrosis in the interventricular septum and posterior wall of the left ventricle, and advanced coronary atherosclerosis.

Case 2. The patient was an 89-year-old woman admitted to the Annie Warner Hospital on February 7, 1952. Her chief complaint was pain in the right upper quadrant, with fainting and vomiting of several days' duration. A good record of her past medical history could not be obtained. In the dispensary, the blood pressure was found to be 240/70, with a regular pulse rate of 38 beats per minute. While the attending nurse was giving the patient a cleans-

ing enema, her breathing suddenly became labored and noisy. She became pulseless, markedly cyanotic and suffered a convulsive seizure with loss of consciousness. After sixty seconds respirations improved, her face became flushed and several seconds later consciousness returned. On further examination, the patient, a small frail person, was found to be dyspneic, with distended cervical veins and thickened, hard arteries at the wrists. There was bilateral arcus senilis. The chest was emphysematous, with physical signs of a moderately large effusion in the right thorax, and basal crepitant rales. The legs were markedly edematous to the knees. Examination of the heart disclosed an apex beat to the left of the midclavicular line in the fifth interspace. There was a highly pitched systolic murmur at the apex, which was transmitted to the axilla; in the aortic area, a rough systolic murmur was heard, transmitted to the neck vessels. The abdomen was markedly distended, with a tender liver edge palpable below the rib margins. Peripheral arterial pulsations were not detected in either foot. Neurologic examination gave normal findings.

The results of laboratory studies were as follows: Urinalysis—albumin, 4 plus; specific gravity, 1.017; 5–10 hyaline and granular casts with an occasional RBC per LP field. The Wassermann reaction was negative. The blood urea nitrogen level was 38 mg. and creatinine, 1.65 mg. per 100 ml. Hemogram—RBC, 4,120,000 per cu. mm.; WBC, 11,500 per cu. mm.; and Hb, 11.5 grams. EKG showed a complete heart block with a ventricular rate of 38; the QRS-interval was .06 sec., and the Q-T interval, .56 sec; the S-T segments were depressed in L_1, and V_{4-6}; the T-waves were inverted in L_{1-2} and V_{3-6}.

The patient was rapidly digitalized. On the third hospital day, 750 cc. of clear amber fluid was removed from the right pleural space. On February 19, 1952, after she became edema-free, she was started on an ambulatory routine. On February 23, after eating breakfast, she was found unconscious in a convulsive seizure. She was incontinent and had vomited. The pulse was not perceptible during the attack, which lasted for five minutes. From February 23, 1952, to March 8, 1952, while bedfast, she suffered 15 separate attacks of unconsciousness, with duration varying from thirty seconds to five minutes. In many of these seizures, sharp thumping with the fist over the precordium tended to terminate the attack. On two occasions the pulse was detected at 68–70 per minute, only to return to 36–40 when the patient regained consciousness. From March 8 to May 8, 1952, she was fully compensated and was free of all attacks. She refused to take heavy foods or to swallow capsules of Pronestyl, complaining that these procedures would cause her illness. During this time she was walking daily increasing distances with comfort. Towards the end of this two-month period, she again exhibited signs of left ventricular failure with basal rales and signs of collection of fluid in the right pleural space. On May 8, 1952, she experienced loss of consciousness in a convulsive seizure with imperceptible pulse, which lasted four minutes. Although 475 cc. of clear fluid was removed from the right pleural space and she was again kept bedfast, she experienced 6 separate attacks over a 35-day period. The duration of the attacks varied from forty seconds to four minutes. She died suddenly in an Adams-Stokes attack on June 13, 1952. No autopsy was obtained.

DISCUSSION

As observed in Case No. 1, the occurrence of an Adams-Stokes attack is often mistaken for a "little stroke" or a psychomotor seizure. It is only a repetition of these attacks in a patient with bradycardia that finally indicates the correct diagnosis. It is strongly recommended that every patient (in the geriatric group particularly) found to have a persistently slow pulse be subjected to a complete electrocardiographic study, in order to determine the presence or absence of a heart block. It must be noted that Adams-Stokes attacks may occur with paroxysmal heart block complicated by ventricular arrhythmia. They have also been reported to occur more frequently in patients with partial heart

block (23). The period of sudden change from a partial to a complete heart block is the time when the heart is most susceptible to ventricular standstill.

Not every case of complete heart block will ultimately result in Adams-Stokes disease, as shown by the work of Cowan and Ritchie (24). These workers reported in 1935 that Adams-Stokes disease developed in 33 per cent of 78 cases of complete heart block. As suggested by Schnur (22) myocardial anoxemia, with its deleterious effect on the A-V node and bundle of His, may provide a plausible explanation. He postulated, after studying a case which exhibited three different mechanisms precipitating the attacks, that in areas of the myocardium (possibly adjacent to the junctional bundle) there exists a relative or at least a potential myocardial ischemia due to coronary sclerosis. Many physiologic factors affecting coronary blood flow, such as increased exercise (which causes no appreciable clinical effect in normal individuals, because of adequate reserve), grossly exaggerate the coronary insufficiency and precipitate an attack of irritative myocardial anoxemia. Riven, Blake and Thomison (6) recently reported a case of ventricular standstill with multiple ectopic arrhythmias. At autopsy there was histologic evidence of coronary atherosclerosis involving the small intramyocardial radicles of arteries; this had resulted, over a period of years, in multiple tiny infarcts in the region of the A-V node and the bundle of His. To these lesions the authors attributed the intermittent conduction defects and terminal ventricular tachycardia. Conceivably, in patients with chronic rheumatic heart disease and damaged junctional tissue, superimposition of coronary sclerosis (producing over the course of years anatomic changes such as Riven *et al.* have described) could jeopardize normal rhythm under the stress of exercise, and thus predispose to Adams-Stokes attacks. Such a condition was found at autopsy in Case No. 1, the 83-year-old woman, who suffered repeated attacks during small increments of exercise, such as attempting to sit up and swallow food or moving from one side of the bed to the other. The role of anoxemia, likewise, is illustrated in Case No. 2, the 89-year-old woman, who experienced repeated bouts of Adams-Stokes attacks, associated with the development of a moderately large hydrothorax. The decrease in pulmonary reserve produced by the collection of fluid in the right thorax, when superimposed on the senile emphysema and advanced atherosclerotic cardiovascular changes, may have produced irritative myocardial ischemia. In a case of this type, the presence of marked cerebral arteriosclerosis could be a contributory factor in the symptom-complex of dizziness or vertigo with third-degree heart block. It was interesting to note that after overcoming decompensation and removal of the pleural effusion this 89-year-old patient became ambulatory, and had no Adams-Stokes attacks until left ventricular failure again set in.

Although in smaller hospitals, the reduction in laboratory and nursing personnel makes it difficult to obtain on-the-spot electrocardiograms, every effort should be given to obtain tracings before and during the attacks, in order to establish the mechanism. This is important for two definite reasons: 1) diagnosis and 2) treatment.

Cardiac syncope of neurogenic origin is differentiated in that the auricles do

not contract; it is a sinus-node asystole. In Adams-Stokes attacks, on the other hand, the auricles do continue to contract. In analyzing the 36 cases of complete heart block associated with attacks of unconsciousness as reported by Parkinson, the sex differentiation is interesting as regards the mechanism which set off the attack. In complete heart block with sudden onset of ventricular standstill, the incidence was predominantly in males (94 per cent). In complete heart block with the onset of ventricular standstill preceded by ventricular tachycardia, the incidence in males was about equal to that in females. In complete heart block without ventricular standstill and with the sudden onset of ventricular tachycardia and/or ventricular fibrillation, the incidence was predominantly in females (75 per cent). Does the higher incidence of atherosclerosis in the male with anoxemia have any significance with regard to this entity? In the 2 cases reported in this communication, it is conjectured that ventricular arrhythmias with or without ventricular standstill caused the attacks. This suggestion is prompted by the duration of the periods of unconsciousness. Schwartz (25) has stated that a loss of consciousness for a period longer than three minutes is likely to be due to ventricular tachycardia and/or fibrillation.

The treatment of the cardiac emergency produced by the Adams-Stokes attack is dependent on the mechanism operating in each case. In pure ventricular standstill, the emergency treatment of choice is intracardiac adrenalin. If ventricular tachycardia and/or ventricular fibrillation is the mechanism setting off the attack, the treatment of choice is quinidine or Pronestyl. Paul White warns against the use of adrenalin in the presence of a ventricular tachycardia because of the possibility of its stimulatory effect on fibrillation. In Case No. 2, the simple procedure of sharp thumps over the precordial area was successful in aborting some of the attacks.

In the course of this disease, the better prognosis can be given to the cases exhibiting pure ventricular standstill. In the cases exhibiting ventricular tachycardia and/or ventricular fibrillation there is usually no chance for recovery. Patients in this category rarely survive a year from the time of the initial attack. Campbell (21) has estimated from his study of this disease that two thirds of these patients die in an Adams-Stokes attack. The rest succumb to coronary occlusion with myocardial infarction, or to congestive heart failure.

SUMMARY AND CONCLUSION

Two cases of Adams-Stokes disease are reported, occurring in women 83 and 89 years of age, respectively. It is postulated that with the increasing size of the geriatric age-group the incidence of Adams-Stokes disease will increase. Establishment of the mechanism of the attack can be made only with the application of electrocardiography. The importance of knowledge of the mechanism with regard to diagnosis, treatment and prognosis is discussed.

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GOUT

A Summary of Recent Developments in Therapy*

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One of the most important contributions of recent researches in the field of gout has been the work of clinicians who have at last clearly delineated the extended biologic course of this progressive ailment. A much better understanding of the disease has resulted and because of this work, physicians are now in a position to make the diagnosis of gout promptly even during the early stages, whereas formerly its recognition was very difficult. Search for better therapeutic measures with which to influence the course of gout has received strong impetus from this improved understanding of the disease.

In brief, these studies have disclosed that symptoms of gout progress through an initial or larval period, which generally is symptomless, a second period of acute articular attacks, and a third chronic period marked by progressive articular deformities and destruction of certain internal organs by tophaceous deposits. For some individuals with gout, the final period is never reached because of slow development of the evolutionary process or because of effective treatment during earlier stages of the ailment.

The larval period is the time prior to the appearance of gouty arthritis. This is the period during which the biochemical foundation is being laid within the organism, and on this foundation the later symptoms are based. The larval period may begin *in utero* for those individuals who inherit the gouty disorder. For others, inception of the larval period occurs during the early years of life. The larval period may range in duration from a few years for some individuals to half a century or more for others.

Advent of articular attacks may be looked upon as marking a transition from the initial period to the second period of gout. During this second period, articular attacks ordinarily behave in a characteristic manner and differ notably from articular symptoms of all other forms of joint disease. Second-period attacks of gout appear usually without premonition and usually affect the articulations of the distal portions of the extremities. After a few days, such attacks subside without residual deformity.

After a number of years, some gouty patients experience a gradual alteration of symptoms, and the appearance of these altered symptoms may be considered to mark the opening of the third period—that of chronic gouty arthritis. Non-remitting painful articular deformities characterize this period. These reflect a progressive destruction of osseous and ligamentous structures by gouty lesions. Once acquired, such deformities are nonreversible and may be expected to progress gradually, causing increasing disability as time passes.

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It is during the third period that visceral lesions are apt to determine the clinical picture. During this phase renal lesions are often rapidly progressive, causing more or less severe arterial hypertension in some patients, and albuminuria with nitrogenous waste retention in others. Arteriosclerosis is often present during this third period and for many patients with gout, appears to have an accelerated course. Life may be terminated prematurely by occlusions of coronary or cerebral arteries. During the third period, subcutaneous deposits of uric acid may increase in size to extraordinary proportions. Continuing centrifugal growth of these masses may destroy overlying integument in numerous places, resulting in multiple sinuses which discharge necrotic material containing sodium urate.

COMMENTS ON THE PATHOLOGIC PHYSIOLOGY OF GOUT

The fundamental problem of what causes gout remains unsolved, although some aspects of the abnormal biochemical processes which are at work in gouty individuals are being slowly elucidated. Researches with radioactive isotopes have played a considerable role in these developments, and physiologic studies with cortisone and adrenocorticotropic hormone have also helped to solve some of the problems in this field.

The cause of gout must certainly have something to do with the metabolism of uric acid, inasmuch as this substance appears everywhere among the pathologic lesions of gout, and relatively large amounts of uric acid tend to accumulate in circulating fluids of gouty patients. The basic mystery has to with the question of why so much uric acid is produced or retained by the gouty patient and by what mechanism his uric-acid production or excretion differs from normal.

Until recently it has been accepted that uric acid in man is an end product of nucleoprotein metabolism and that no other source gives rise to uric acid in the human organism. However, studies with isotopic tracers appear to have thrown a different light on this subject. By administering a number of labeled compounds, new information has been obtained which seems to indicate that uric acid in man may come not only from breakdown of nuclear material but may in fact also be formed by building up relatively simple compounds into the more complex uric acid molecule (1).

The nucleus around which other carbon and nitrogen atoms are associated for this biosynthesis appears to be glycine. However, lactic acid, acetic acid and formic acid, all derived from metabolism of carbohydrates, join in this synthesis to form the ultimate molecules of uric acid (2).

This information may in time prove of greatest importance from the standpoint of treatment for this disease, inasmuch as efforts to influence the course of the ailment by limiting the intake of purine-containing foods, that is, foods rich in nuclear material, may need to be revised. We may be barking up the wrong tree, so to speak, by stressing the importance of restricting the intake of meat and glandular substances; perhaps very different procedures will be found for preventing the accumulation of uric acid.

Curiously, it is not yet known with certainty that all gouty patients do indeed suffer from an increased rate of production of uric acid from precursors. That is to

say, this work has not yet proved that all gouty patients suffer from making too much uric acid because of a specific biochemical speed-up of this particular set of reactions. In fact, evidence on this point appears to indicate that uric-acid production in gouty subjects may be no more rapid or efficient than in normal individuals (3).

If then, the gouty patient does not produce more uric acid than normal, what can possibly account for retention of this substance in his blood and accumulation in bones and joints? The answer does not lie in any decreased ability to excrete, for gouty patients may excrete uric acid with the same ease as normal persons. Uric-acid clearance rates for the kidney may be as high in gouty patients as in normal persons. Moreover, ability to concentrate uric acid by the kidney is not defective in gout, unless renal disease has complicated the picture and has added new, secondary disturbances to this already intricate problem.

A final solution of this enigma is not at hand, although with new techniques, investigations may perhaps progress more rapidly. It seems likely that satisfactory clinical control of gout will have to await the advent of this solution.

A further development of importance in this field is disclosure of existence in the human, of a "pool" of uric acid (4), defined as a quantity of uric acid distributed throughout the organism, capable of mixing promptly with new-appearing uric acid. Newly formed uric acid is being constantly added to this pool from the metabolic mill and it is from this pool that uric acid is removed for excretion through the kidneys. Uric acid accumulated in tophi may be at least in part in equilibrium with uric acid in circulating fluids. Thus the level of blood uric acid may rise or fall and tophi may be caused to grow in size or diminish, depending upon the direction of movement of uric acid either into or out of the pool. Administration of drugs which hasten excretion of uric acid, such as salicylates or cinchophen, may temporarily alter the size of the pool or increase speed of turnover. Studies in progress, and only recently reported, have indicated that the uric-acid pool is far larger in gouty patients than in normal persons, a phenomenon as yet unexplained. The concept of a uric-acid pool has obviously provided a new and perhaps very important intellectual tool with which to implement thinking and facilitate research in this field.

Individuals with gout differ from normal persons in regard to the rate at which certain steroid compounds are excreted in the urine. This distinction concerns especially those steroid compounds having an oxygen atom attached to the carbon atom numbered 17 in the steroid ring structure (so-called 17-ketosteroid compounds). In gout, excretion rates for these compounds are notably less than normal; and inasmuch as these compounds are presumed largely to be manufactured in the adrenal cortex, gout and abnormal adrenocortical function may in some manner be associated. This association is not proved, however, and the concept has not as yet led to a satisfactory treatment for gout, based upon replacement of insufficient adrenal products (5).

Thus are cited only a few trends of current researches in relation to the pathologic physiology of gout. The cause is undisclosed, the basic anomaly is unknown, and a clearly marked direction has not been found to show the proper path of

treatment. Methods in use today reflect properly the results of generations of clinical experience; and, although these therapeutic measures often leave much to be desired, their use may be expected to produce worthwhile results for many sufferers. It is with these methods, largely empiric and time-proven, that further comments of this paper will be most concerned.

THE TREATMENT OF GOUT

General comments on treatment of gout

Success in overcoming acute attacks does not necessarily imply a successful control of the underlying disorder; neither should it be assumed that the responsibility of the physician has been discharged when the acute attack has subsided. Consideration should be given in every instance, not only to treatment of the acute attacks but also to the possibility of influencing the course of later events so as to reduce the speed of progression of the disease and, if possible, to bring it to a halt.

If this goal is to be achieved, certain requirements should be fulfilled to the best possible degree. In the first place, the condition of each patient should be properly assessed as regards the state of the disease and the presence of important complications. Subsequently, the patient should be informed as to the nature of his ailment, its potential importance as regards health and longevity, and the methods presently available for its control. To accomplish these purposes, thoughtful discussions between patient and physician are desirable. Wholehearted cooperation of each patient should be sought at the outset, just as the physician seeks cooperation and understanding of patients suffering with diabetes mellitus in order to achieve efficient management.

Therapeutic programs for patients with gout should be adjusted to suit the situation of each patient. Measures which are appropriate for one individual may not suffice for the next. A program adequate for the larval period does not suffice to handle the more serious difficulties experienced by patients during the second and third periods of the disease. In a sense, therefore, therapeutic programs for patients with gout should be tailor-made, and the following general rules should be adapted to the changing problems created by this many-faceted disease.

General principles of treatment for the larval period

If gout is to be discovered during the larval period, physicians should examine carefully the family members of affected patients, testing blood uric-acid levels from time to time and making careful inspections for early signs of subcutaneous tophi. If the diagnosis of larval gout is established, the patient should be instructed to eat in moderation of foods containing purines, to avoid alcohol, to choose occupations which do not encourage excesses of eating, and to avoid occupations which involve exposure to lead.

An incident of articular pain occurring in a patient during this period should call for a careful review of the situation. If it is determined that the patient has progressed into the second period of acute articular attacks, more intensive regimens should be instituted, as outlined in the section which follows.

Treatment during the period of recurring acute gouty arthritis

Treatment during this period should require two sets of instructions. One is to be followed while the patient is free of articular pain, and one is to be used for combatting acute attacks which occur in spite of interval treatment. Attack-free intervals should be looked upon as periods during which the patient is in danger of experiencing further attacks.

The aim of treatment during free intervals should be to restrain the progressive tendencies of defective metabolism of uric acid.

Purine-containing foods should be allowed only in moderation. Protein-containing foods which are low in purines (including particularly milk, cheese, eggs and breads) may be substituted for meat. Bulk, vitamins, and mineral requirements are provided by vegetables and fruits. Caloric needs should be taken into account.

Meats are permitted in limited amounts and glandular meats, including sweetbreads, liver and kidneys, are excluded entirely. Certain vegetables and cereals contain notable amounts of purines, and therefore should be permitted only sparingly. Asparagus, cauliflower, kidney beans, navy beans, lima beans, onions, peas and spinach, although not rich in purines, nevertheless contain amounts sufficient to justify their general avoidance by the worst-affected patients and their use in strict moderation even by those who have the disease in its milder forms. Whole-grain cereals, whole-grain bread, and malted cereals should be used in the same manner. Soups made of meat stock and gravy contain notable amounts of purines, hence should be avoided.

A workable arrangement of the diet for attack-free intervals is as follows: During two or three days of each week, foods containing significant amounts of purine should be omitted entirely. For a patient with gout of "average" severity, a small serving of nonglandular meat may be allowed on three days of each week. Other purine-containing foods should be omitted on these days. Either one serving of a purine-containing vegetable such as cauliflower or spinach, or a serving of whole-grain cereals such as cooked whole wheat, wheat biscuits or oatmeal, may be allowed on two additional days of each week in such "average" instances. In very mild instances, a serving of meat may be permitted daily; but in more severe cases, only one or two meat servings weekly.

Foods permitted freely in this diet include refined cereals (cream of wheat, corn flakes and puffed rice) and white bread or fine rye bread, which should be used in place of whole-wheat bread.

Uric-acid levels of plasma tend towards higher concentrations in patients permitted free intake of fat-containing foods, and lower levels are encouraged by high-carbohydrate diets; hence butter, cream, mayonnaise and other fats should be included only in moderate amounts. The caloric value of eliminated fat should be replaced by carbohydrate foods.

For desserts, fruits are satisfactory. Ice cream, tapioca, cornstarch pudding, sherbets and ices may be permitted for patients who are not overweight. Desserts to be avoided include those rich in fats, chocolate cake or pudding, spiced cake, cookies, pies, and ice cream made with rich cream.

Medications during intervals of freedom

Weekly courses of salicylates, 75 to 90 grains (5 or 6 grams), on three to five consecutive days of each week stimulate excretion of urates. In order to avoid crystallization of urates in urine during urate diuresis, sodium bicarbonate may be given on days when the salicylate diuresis is in progress, in amounts sufficient to alkalinize the urine.

Treatment of acute attacks

Rest for affected joints: If an attack occurs, the affected extremity should be elevated; and in all but mild episodes, complete rest should be continued until swelling and tenderness have cleared. Premature return to activity may be followed promptly by recurrence of the attack.

Application of heat: Hot compresses may be effective for alleviating pain. To prevent maceration by the wet cloths, the skin should be smeared with vaseline or cold cream. Compresses should be large enough to extend well beyond the painful region. A thin blanket wrapped about the wet compress reduces speed of cooling. Compresses should be renewed at intervals of one to two hours, and this process should be continued until the attack is controlled.

Colchicine: This ancient remedy is now available in potent form as tablets or pills of $\frac{1}{100}$-grain or $\frac{1}{120}$-grain strength (0.65 or 0.53 mg.). A "course" of colchicine, *i.e.*, the number of tablets which suffice to induce nausea or diarrhea, should be given at the outset of an acute attack. A satisfactory method of employing colchicine consists in administering two tablets at the start, followed by one every hour or two, stopping when diarrhea appears. On the average, it takes from six to fifteen doses to induce diarrhea.

The mode of action of this remedy remains obscure. Administration of the maximum tolerated amount has no effect on the level of uric acid in plasma and provokes neither increase nor decrease in the rate of excretion or renal clearance of uric acid. Nevertheless, the effectiveness of colchicine against acute attacks of gout is notable in most instances. One writer recently reported that relief of acute attacks occurred in more than 95 per cent of individuals, following proper administration (6). In occasional instances colchicine fails to relieve the pain of gout. A second or even a third course may be employed for these stubborn attacks, allowing an interval of a day or so between each course.

Comments on the Prophylactic Use of Colchicine During Intervals Between Attacks: Employment of colchicine during intervals between attacks has found favor with some students of gout but is not approved by others. Those who favor administration for prophylactic purposes do so because of an impression that attacks occur less frequently with this regimen. However, decisive proof of its value has not been forthcoming; and for the present, the writer does not employ interval administration of colchicine.

Comments on Intravenous Administration of Colchicine: Colchicine may be given intravenously (although preparations for intravenous administration are

not yet generally available in this country). Ordinarily the drug is effective when given by mouth; hence, intravenous use is not often necessary. For occasional persons unusually sensitive, diarrhea and nausea make oral administration of this drug difficult, and for such persons intravenous therapy may be useful. Acute attacks of gout may respond satisfactorily and no unusual reactions have been observed with this mode of administration (7).

Diet: The diet should be as free of purines as is practicable while an acute attack is in progress. Also, careful attention should be given during attacks to avoidance of fat-containing foods and to supplying generous amounts of carbohydrates. Milk, cheeses, eggs, cereals, vegetables and fruits should constitute the chief categories of food for this period. Vegetables which contain even moderate amounts of purines should be avoided during acute attacks although coffee, tea and chocolate need not be eliminated.

Other medications: During acute attacks, sodium salicylate may be administered in doses of 75 to 120 grains (5 to 8 grams) daily. In especially severe or stubborn episodes which resist these medications, this writer employs cinchophen in doses of 7½ grains (0.5 gram) three times daily. In this regard it may be noted that hepatitis following administration of cinchophen for treatment of gout has not been reported.

Narcotics: During acute attacks of gouty arthritis, pain may be intense and may necessitate administration of a narcotic. Codeine, ½ to 1 grain (30 to 60 mg.) at intervals of three to four hours; or Demerol, 50 to 100 mg., may be employed.

Comments concerning incidents which may provoke onset of acute gouty arthritis

A number of physical, occupational, pharmacologic and psychologic stimuli have been identified as provocatives of acute attacks of gouty arthritis in susceptible persons. Avoidance of these incidents constitutes a most important therapeutic measure for gout.

Among incidents which may provoke attacks of gout are the following: 1) wearing of tight-fitting shoes or rapid breaking-in of stiff, new shoes; 2) prolonged automobile driving; 3) excessive walking or standing; 4) unusual exertions connected with sporadic participation in sports or hobbies; and 5) taking of certain pharmaceutical agents, including bile salts, concentrated solutions of thiamine chloride, liver extracts, strong diuretic agents and purgatives.

In the event that gout is associated with diabetes mellitus, particular care should be taken to avoid insulin reactions, as acute gouty arthritis may follow promptly after episodes of hypoglycemia.

Many major and minor surgical procedures provoke exacerbations of gouty arthritis unless the patient is prepared. Such postoperative attacks may be prevented if, during a period approximating ten days prior to surgery, the patient takes only purine-free foods, and also full doses of salicylates or cinchophen. On the day of surgery, and for a week or ten days thereafter, this program should be continued.

Comments on therapy of the special problems encountered during the third or final period of gout

During this advanced state, acute attacks may be expected to occur with increasing frequency; and continuing, but less severe pain may persist between attacks.

For severe episodes, treatment should follow the principles just outlined for attacks during the second period. Analgesic drugs, regular rations of salicylates, and meticulous adherence to dietary restrictions are fundamental to control of interval symptoms during the third period.

For advanced articular deformities caused by enlarging tophaceous deposits, surgery may provide notable relief. Possibilities of surgical treatment for this stage of gout have only recently been studied and the indications are not widely appreciated. If surgery is undertaken, removal of tophaceous material should be as complete as possible, even though sacrifice of important osseous or ligamentous structures may be required. Use of drainage tubes and rubber dams add little or nothing to speed or smoothness of recovery from these operations, hence should be avoided whenever possible. After excavating all visible tophaceous material, overlying skin should be approximated with loose sutures and dressings carried out as for any other surgical wound.

Suitable for surgical removal are tophaceous deposits large enough to interfere with function of joints, especially if accompanied by ulceration of overlying skin, with discharging or draining sinuses. Chronically enlarged ulcerating bursae over elbows, knees or other bony prominences may also be removed. At times these may be "shelled out," thus permitting a clean and complete removal.

Treatment for complications resulting from arteriosclerosis during advanced stages of gout

This should follow principles appropriate for treatment of arteriosclerotic ailments in non-gouty individuals. Coronary artery disease and severe arterial hypertension require a mode of life which avoids undue physical and psychologic strains.

Cerebrovascular accidents or strokes are not at all uncommon during this period and are handled in the usual manner. Myocardial infarction is often a cause of disablement and frequently a cause of death among gouty patients. Renal deterioration requires separate and special measures appropriate to the disturbances of physiologic function resulting from this complication, inasmuch as renal lesions in gouty patients do not always have the same underlying pathology. In some patients, invasion of renal parenchyma by gouty granulomata or tophi is responsible. These tophi may enlarge and multiply, destroying large portions of the renal substance. In others, renal deterioration results from chronic fibrosing changes similar to those encountered in advanced glomerulonephritis or severe arterial hypertension. Still others have renal damage caused by pyelonephritis. In some gouty patients renal symptoms are caused by heavy accumulation of uratic crystals. These may descend into the ureters, giving rise to symptoms and complications characteristic of renal calculi.

Comments on some "newer" agents for the treatment of gout

Cortisone and adrenocorticotropic hormone: Among effects produced by these hormones are alterations in the blood level and excretion rate of uric acid. A relative increase occurs in the amount of uric acid excreted in urine, as compared to other nitrogenous waste products. In some instances this change is accompanied by a lowered level of blood uric acid and by a diminution in size of the "miscible pool" of urates.

As regards administration of these hormones in the treatment of gout, results may be summarized as follows: Acutely painful and swollen joints may show notable improvement within a few hours or after a day or so. This effect is not invariably obtained, however, and failures are as frequent with these agents as with the conservative measures already noted. In acute gout, the abrupt withdrawal of therapy with cortisone or of adrenocorticotropic hormone may be followed by prompt return of symptoms. In order to prevent such recurrences two procedures have been recommended: a) gradual, rather than abrupt withdrawal of hormone, and b) administration of colchicine in connection with, and for several days after the hormonal treatments. Both procedures may fail in some patients, and the whole problem of hormonal therapy for gout needs more study.

Experiences with hormonal treatment of chronic gouty arthritis have been limited; hence, it is not possible at this time to draw any conclusion about the value of such long-term therapy. Partial regression of subcutaneous tophaceous nodules has been reported, and also a lowering of blood uric acid levels and increased uric acid excretion. Acute attacks have occurred in spite of long-continued administration (8).

For the present, the author reserves the use of cortisone and of adrenocorticotropic hormone for severe and stubborn attacks of gouty arthritis which resist ordinary measures of treatment.

If hormonal therapy is undertaken, either cortisone or adrenocorticotropic hormone may be used, as each is effective. Adrenocorticotropic hormone may be administered in doses of 50 mg. intramuscularly at intervals of six or eight hours during the first twenty-four hours. Thereafter, 10-mg. or 20-mg. doses are given daily for three or four days. Colchicine is also administered from the outset and is continued until looseness of stools is provoked. After diarrhea is initiated, colchicine may be withheld for twenty-four hours and then resumed in doses of $\frac{1}{100}$ grain (0.065 mg.) three to four times daily for a week or two after the attack subsides.

If cortisone is chosen, an initial dose of 100 mg. should be given, either by mouth or intramuscularly, and repeated three or four times in the first twenty-four hours, as the attack may require. Thereafter a dose of 25 mg. may be administered daily for three or four days. Colchicine may be administered in association with cortisone was in the case of the adrenocorticotropic hormone.

Phenylbutazone (Butazolidin): This substance 3,5-dioxo-1,2-diphenyl-4-n-butyl pyrazolidine is now in process of investigation, and has been reportedly successful in relieving acute gouty arthritis (9). The molecule of phenylbutazone somewhat resembles pyramidon in structure, and may be administered by mouth.

A notable physiologic effect of its administration, pertinent to the problem of treatment for gout, is increased excretion of urinary uric acid and lowering of the level of uric acid in serum.

A final estimate of the value of this compound for the therapy of gout cannot be made at this time. Still to be answered are important questions, including whether this substance is more efficient than colchicine, and whether notable and even fatal reactions already reported (dermatitis, water retention, gastrointestinal irritation, stomatitis, and hematologic disorders including fatal and non-fatal agranulocytosis) will eventually prohibit use of this agent. Perhaps brief administration such as may be required for treatment of acute episodes of gouty arthritis can be carried out without notable risk of reactions. However, further study is required before a decisive answer can be expected.

Probenecid (*Benemid*): This compound (p-[di-n-propyl-sulfamyl]-benzoic acid) has been investigated recently in this country for treatment of gout and has been described as "a potent preparation as well as a drug of low toxicity for prolonged administration" (9). An effective and persistent uricosuric action has been observed. This supposedly results from the alteration of transport of uric acid in such a manner that reabsorption by renal tubules is reduced; hence, excretion is augmented. Excretion rates have been observed which are up to 90 per cent greater than in untreated individuals. Results obtained thus far indicate that acute attacks of gouty arthritis are not relieved by this agent; in fact, attacks may appear while the drug is being administered. Effectiveness in augmenting excretion of uric acid is thought to be of possible usefulness in long-term treatment, but more data are needed on this point also.

Partial regression of tophi has been reported during administration of Benemid.

The toxicity of Benemid cannot be finally assessed because of the limited information. Some of the reports indicate a need for further careful study of the problem.

SUMMARY

The stages of gout, its pathologic physiology and treatment are discussed, including a critical appraisal of the most recent developments.

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CUTANEOUS NEW GROWTHS OF GERIATRIC INTEREST

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There are several new growths affecting the skin which are not well known to many practitioners. There is some confusion concerning their potential for malignant degeneration. In this category fall the senile and the seborrheic keratoses.

Senile keratoses undergo cancerous change in from 20 to 25 per cent of cases, whereas seborrheic keratoses, although reported as becoming cancerous on rare occasions, may for all practical purposes be regarded as entirely benign.

It is common experience, however, for the dermatologist to have referred to him patients with seborrheic keratoses who are in a state of apprehension because they have been informed that the lesions must be removed at once. Such instruction to the patient usually results from the physician's failure to make a distinction between the senile and sebaceous types of keratosis, or because the frequently blackish color of the latter type suggest the possibility of melanoma.

It is understandable that confusion may exist in the mind of the physician who does not have a special interest in these lesions, since for many years there was no clear distinction between the two conditions. Becker (1) recently listed 23 names which have been applied to seborrheic keratoses between 1864 and 1948, and the terms senile keratoma and senile acanthoma have been used as synonyms for senile keratosis. As 4 of the names used to designate the seborrheic variety include the term senile, errors in interpretation are to be expected. From 1926 on, however, several writers have clarified this picture (as in a recent article by Caro and Szymanski (2)) until it is now accepted that we are dealing with two separate entities. In the vast majority of cases the experienced observer is able to differentiate them on purely clinical grounds, and only rarely is it necessary to resort to histopathologic interpretation of the nature of the lesion.

SENILE KERATOSES

These lesions are seen most often in persons in the later years of life, but may be seen in younger people, particularly in those with a blond type of skin, and those whose skin has been unduly exposed to the sun. Kennedy (3) states that senile keratoses are seen most frequently in patients past 40 years of age; and as in the case of basal-cell and squamous-cell epithelioma, the incidence doubles after the age of 60 years.

The condition is seen more frequently in males than in females, as males usually are more exposed to the elements and are less likely to resort to the use of lubricating creams and protective lotions or unguents. Blonds and persons with fair skin are affected much more frequently than those with dark skin.

As is to be expected, these keratoses occur, as a rule, only upon the exposed

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parts of the body, so they are met with most frequently on the face and dorsal surface of the hands, and less often on the neck and forearms.

The skin of the exposed parts of persons showing lesions of this type usually has undergone some degenerative changes before the keratoses make their appearance. Eller (4) has stated that senile keratoses are so commonly associated with senile atrophy of the skin that this atrophy may be considered a part of the syndrome. The senile skin is thin, the sclerosed vessels cause a lessening of the blood supply, and the elastic fibers and collagenous tissue undergo degeneration. At the same time the hair follicles and the hair and the sweat glands become atrophic, so that moisture and oil are decreased and the keratin produced is dry and more adherent. The skin changes from its normal red or pink color to a yellowish or grayish tint. Telangiectases and freckles are often present.

On such a foundation a varying number of senile keratoses may develop. At times only a single lesion is present, but usually the lesions are multiple. In people who have spent much time out of doors there may be a very large number of keratoses, and in earlier days the term "farmer's skin" or "sailor's skin" was used to designate those so severely affected. In this country the incidence of senile keratoses is much greater in Texas and in the southwestern states than in other parts of the country. Keratoses may develop, however, in persons who have spent most of their time indoors, but who nevertheless show the cutaneous senile changes described previously.

The characteristic senile keratosis is at the outset well demarcated; it is irregular or (less often) round in outline, flat or slightly raised, and commonly with an erythematous base. The color of the lesion, which is due largely to the covering scale, may be grayish, yellowish, brownish or grayish black. The scale is dry, hard and very adherent; it may be depressed at the center or may be heaped up to present a horny mass, varying in thickness in different parts of the lesion. When this scale is removed by curettage, a sharply defined bleeding erosion is seen. Patients commonly state that in the earlier stages the scale would come off spontaneously or while washing the face, only to reform after a variable period of time. After repeated occurrences of this type, the scale becomes persistent.

Senile keratoses as a rule evolve slowly, but tend to become larger gradually, while new lesions make their appearance. Certain of the lesions become frankly verrucous, showing more or less prominent horny elevations; in some instances there is formation of true cutaneous horns.

When malignant degeneration begins to take place, certain alterations usually mark the change in the character of the lesions. The development of inflammatory changes, if not due to infection (rare), should arouse suspicion. An inflammatory areola appearing about the lesion is especially significant, as is replacement of the scale by a crust. Rapid increase in size or elevation, an increase in the erosive or ulcerative element, an increase in the number of telangiectatic vessels, and the appearance of a pearly border in the lesion all suggest the probability that cancerous change is supervening. When cancer develops in senile keratoses it is

usually of the prickle-cell type, but has been reported as being of the basal cell type in rare instances.

Despite the fact that seborrheic and senile keratoses may resemble each other clinically, their microscopic characteristics are usually different, and an experienced pathologist should have little or no difficulty distinguishing them.

In senile keratoses a markedly thickened scale containing parakeratotic nuclei is easily discernible. The malpighian layer varies in thickness. Atrophy and hypertrophy occur in alternate zones. The normal differentiation of cells from the basal layer through the prickle layer to the granular layer is lost, resulting in a disorderly arrangement of the epidermal cells. The nuclei appear hyperplastic and hyperchromatic throughout the epidermis. In addition, one sees such atypical features as dyskeratotic cells, mitotic figures and vacuolated cells. The basal layer is intact, signifying that the lesion is still benign. However, when a diagnosis of senile keratosis has been made microscopically, serial sections should be made, in order to be certain that the lesion has not broken through the basal layer, with the production of an epidermoid carcinoma in another area.

Senile keratoses may be differentiated from seborrheic keratoses microscopically by fact that in the seborrheic variety there is a basal type of cell, upward growth, and horn-cyst formation.

Treatment. From the standpoint of treatment, it is advisable to warn the patient about further exposure to sun and wind, insofar as possible. Bland oils or cream should be used regularly to overcome the dryness of the senile skin.

Selective destruction of such lesions may be carried out effectively by a number of agents. Curettage followed by electrocautery, electrodesiccation, electrocoagulation or the cutting current is preferred by some, whereas others prefer to use one of the latter procedures, following this with curettage in order to make certain that all of the diseased tissue has been removed. Destruction may be accomplished by means of freezing with carbon dioxide snow, or the use of acids such as mono- or tri-chloracetic acid, especially in older patients. Healing usually takes place readily, except in areas where it may be retarded by friction or motion of the parts. In some cases surgical removal of the lesion is to be preferred. The use of x-ray or radium irradiation in the treatment of superficial senile keratoses is not advisable, because the results are inconstant and because the affected skin has already shown change as the result of exposure to actinic energy.

Many lesions which at the time of treatment appear insignificant, may later undergo changes which make their removal advisable. Therefore, patients with multiple lesions should report for observation from time to time.

Recently there have been reports in the literature suggesting that vitamin A may be of benefit in the control of this condition.

SEBORRHEIC KERATOSES

Keratoses of this type may occur at a relatively early age, but are found most commonly from the age of 60 years and upward. They are rare before the age of 30 years, and Becker (1) quotes Pollitzer as stating that they are not seen before the age of 40 years. MacLeod (5) finds that they occur chiefly in elderly people,

but they may occur at an early age in persons with a greasy seborrheic type of skin. Some authorities feel that the incidence in the sexes is about equal, whereas others believe that women are affected more frequently. In our experience, the type of case in which large numbers of the lesions occur on the trunk is met with more frequently in women. Seborrheic keratoses are usually classed as nevi, but they are also attributed to infection, to senile change in the skin, to localized seborrhea and to altered products in the secretion of the cutaneous glands. Becker, who has studied these lesions extensively, regards them as benign epidermal neoplasms.

In seborrheic keratosis the skin does not show any of the clear-cut preliminary changes seen in the case of senile keratosis. The patients are usually in the dark complexioned group, especially those with a greasy type of skin.

The number of seborrheic keratoses which may be present in a given case varies from one or a few, to literally hundreds. The largest number of lesions is usually found on the trunk, especially on the upper part, both anteriorly and, posteriorly. In women the skin beneath the breasts is often heavily involved and lesions about the axilla and umbilicus are often seen. On the face, the temporal region is the site of predilection, and keratoses are often found in the hair line. Thus it is seen that the seborrheic areas are particularly apt to be involved, whereas the extremities are usually spared.

These growths usually begin as skin colored or slightly yellowish, rounded or oval lesions which appear to be very superficial, as though laid on the skin. The surface is flat, or later dome shaped; it may be soft and velvety, or it may be verrucous. The lesions are sharply circumscribed. Their superficial character is indicated by the ease with which the keratosis can usually be scraped off, leaving an oozing velvety surface, sometimes with a verrucous projection or with a honeycombed appearance in the center. As the lesion slowly enlarges it often becomes more verrucous, the greasy scale becomes heavier, and its color becomes darker, often dark brown to blackish. This change is due chiefly to the deposit of pigment, and may give the growth a pseudomelanomatous appearance. The unctuous feeling of the scale is characteristic, and is different from the dry feeling of the scale in senile keratosis. Although the usual lesion is pea sized to coin sized, some lesions enlarge or become confluent to form considerably larger patches. The greasy scale may at times overhang the margins of the lesion, giving it a sessile appearance. On dry parts of the skin the keratoses are usually dry and hard, but in moist areas, particularly in the submammary region, they may form large vegetating cauliflower-like masses.

Follicular plugging, which shows as black dots, is often seen on the surface of the keratosis; it is the result of the presence of keratotic plugs in surface invaginations.

Although the growth of these lesions is slow, the number usually increases with advancing years. There is no tendency to spontaneous disappearance. At times the scale becomes dry and hard and falls off or is rubbed off, partially or completely, but gradually it reappears.

As a rule there are no associated symptoms, unless the keratoses become irri-

tated or affected. Not infrequently, however, itching is a prominent feature when the lesions on the trunk are numerous.

From the standpoint of treatment, there is little reason to remove seborrheic keratoses, except for cosmetic purposes, or because of discomfort when they are present at points of pressure or friction. If they are to be removed, the use of x-rays or radium is contraindicated, since a chronic radiodermatitis is commonly produced if sufficient radiation is used to accomplish destruction. Any of the permissible measures in the treatment of senile keratoses will give a very satisfactory cosmetic result, since the superficial nature of the lesion precludes the necessity of any deep destruction.

The microscopic features of these benign tumors are usually distinctive and diagnostic, even with low-power magnification. Most striking is the fact that the tumors grow upward from the skin, causing the clinical "tacked on" appearance.

The tumor consists of epithelial hyperplasia with interwoven bands of dark basophilic-staining cells, similar to those seen in a basal-cell epithelioma. The interlacing strands show a retiform pattern surrounding islands of normal connective tissue. A thickened scale is located on the surface, and some of the horny material invaginates downward, forming large keratotic plugs. Another characteristic feature is the presence of horn cysts scattered throughout the epidermis. These horn cysts have been mistaken for the "horn pearls" of epidermoid carcinoma. There should be no difficulty in differentiating them, inasmuch as keratinization in the horn cysts is normal, abrupt, and complete, whereas "horn-pearls" are composed of concentric layers of prickle cells progressing to an unusual type of cornification with the granular layer noticeably absent.

Occasionally an abundant amount of melanin occurs in the basal cells; this gives a very dark appearance to the lesions when seen clinically, so that they may be erroneously interpreted as melanomas.

Basal-cell epitheliomas resemble seborrheic keratoses histologically. A differential diagnosis is usually simple, inasmuch as the cells of basal-cell epithelioma occur in large irregular masses which grow downward, invading the corium. The network pattern, so characteristic of seborrheic keratosis, is absent in basal-cell epithelioma.

MISCELLANEOUS BENIGN NEW GROWTHS

There are three benign new growths which are seen very commonly in middle-aged to elderly persons. These are cutaneous tags of the neck, senile angiomata and senile sebaceous adenomata. Although each of these disorders is of importance from the cosmetic standpoint only, physicians are often consulted about them.

Cutaneous tags of the neck

These lesions occur principally on the neck, especially on the sides, but are seen at times about the axillae, and on the face, particularly on the eyelids. They are almost always seen in females, although occasionally males are affected. The number varies greatly from case to case, with some showing a few lesions

only, whereas others show from one to several hundreds. In the most extensive cases, lesions are frequently found on the upper chest as well. These lesions are often looked upon mistakenly as warts, and by some are regarded as fibromas. Pusey (6) remarked that in his opinion they were related to senile degeneration of the skin. According to Tauber (7), brunettes are affected more often than blonds, especially those with an oily type of skin.

Clinically, there are two types of lesion, and in most cases both are present. The first type is a small raised papule of pinpoint to pinhead size, only slightly elevated, dome shaped, sharply outlined, and having at first a normal skin color; this color may be retained, or a yellowish, brownish or even blackish tint may appear.

The second type of growth, which commonly develops from the first, is a small pedunculated soft fibromatous-appearing lesion. Occasionally such a lesion, especially following injury, will drop off; but the usual type does not disappear spontaneously.

The growths appear most often at, or after the menopause. They develop not infrequently during pregnancy, and in such instances usually disappear spontaneously after delivery. These observations have led some writers to believe that the growths develop as a result of some hormonal imbalance.

When destruction of the lesions is desired for cosmetic reasons this may be carried out readily by electrodesiccation or the electrocautery. As only a touch with the instrument is required, large numbers of growths may be removed at one sitting.

Cutaneous tags are classified as papillomas or soft fibromas by the general pathologist. They usually originate from a thin pedicle which flares out to form an oval or round lesion surrounded by a slightly atrophied epidermis. The collagen tissue within the epithelial boundaries occurs in spongy edematous-appearing bundles that are loosely scattered throughout the tumor. Many dilated blood vessels are seen between the connective tissue bundles.

Senile angiomata

Senile angiomata, which have also been called senile ectasia, papillary ectases, papillary varices and De Morgan's spots, occur from middle age onward, being seen most often in the later years. Men are affected more frequently than women. These lesions are usually seen upon the anterior aspect of the trunk, and occasionally are found upon the face and on the proximal parts of the extremities.

At first the lesions are simply red points, but they gradually enlarge to diameters of $\frac{1}{16}$ to $\frac{1}{4}$ inch. They are sharply circumscribed, bright red to purplish in color, and soft to semisolid. They appear clinically as small angiomas, but are made up of tufts of dilated capillaries, according to most observers.

They have no clinical significance, but if the patient wishes to have them removed, they are generally destroyed by a touch with the diathermy point.

The histopathologic examination of senile hemangiomata reveals a more or less circumscribed group of numerous capillaries that are dilated and lined by a single layer of endothelium. The epidermis may be normal. Usually there is some

atrophy of the epidermis with flattening of the rete pegs due to pressure from the dilated capillaries. This type of lesion is readily recognized microscopically and presents no noteworthy problems from a diagnostic standpoint.

Senile sebaceous adenoma

This condition, which had been described in the foreign literature under a variety of names, had escaped description by all but one of the American writers until Nomland (8) in 1930 called attention to it and suggested the now generally used name of senile sebaceous adenomata.

Although these lesions may occur at any age following puberty, they are nevertheless not often seen till after the fourth decade of life. Usually they occur on the forehead, but occasionally on the cheeks. The number varies considerably, ranging from a few to as many as forty or fifty lesions.

The skin of the face of the affected person usually is of the seborrheic type, or exhibits some degree of rosacea.

The lesions appear as chamois-colored yellowish papules which are small and flat, with little elevation above the skin level. They are irregularly round in outline, and frequently show a central umbilication. On close inspection, a number of small yellowish points are seen on the surface of the papule.

A senile sebaceous adenoma is characterized pathologically by the presence of a fairly well circumscribed collection of normal sebaceous glands. These glands arise from a widely dilated lanugo hair follicle, which explains the central pit. There is still some controversy about whether these tumors should be considered as adenomas or as simple hyperplasia of the sebaceous glands. This question is, however, important only from an academic viewpoint and has no practical significance. These lesions are readily destroyed by diathermy or the electrocautery.

SUMMARY

The diagnosis, pathology and treatment of senile keratoses, seborrheic keratoses, and a few miscellaneous benign cutaneous growths, have been discussed.

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SUBMUCOSAL LIPOMA OF THE COLON: CASE REPORT

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The relatively small number of reports of submucosal lipomas of the colon in the literature warrants the addition of the following case:

CASE REPORT

H. E. W., a 75-year-old white male complained of diarrhea and cramping for three weeks. He had noted nausea, vomiting, bloating and melena. His appetite had been only fair. He denied weight loss. Physical examination revealed an elderly white male in a good state of nutrition. There was a slight splinting of both recti muscles. No palpable masses or free fluid were noted.

Laboratory findings: The leukocyte count was 7,850 per cu. mm., the erythrocyte count 5,000,000 per cu. mm., and the hemoglobin 14 grams per 100 cc. Serologic tests for syphilis gave negative results. Urinalysis revealed a trace of albumin.

X-ray findings: Radioscopic studies after a barium enema showed rapid filling to the proximal third of the transverse colon, where a smooth oval defect was noted (Fig. 1). Post-evacuation studies showed an intussuscepting polypoid lesion (Fig. 2).

Operative procedure: An intussusception was found in the right side of the transverse colon. There was no adenopathy in the mesentery. Within the bowel, a tumor mass was palpated. Traction on this mass dimpled the bowel wall at its point of origin. A 4-centimeter tenial incision was made, and a pedunculated mass was delivered and removed. Recovery was uneventful.

Surgical pathology report: The gross specimen (Fig. 3) measured 4 by 3 by 3 centimeters. The mucosa was glistening and pinkish-gray, except for reddish-brown ulceration over the distal tip. The cut surface (Fig. 4) presented a well demarcated, golden-yellow tumor mass replacing the submucosa. Recent hemorrhage was present at the point of surgical interruption. Microscopically, the tumor mass was composed of mature fat cells.

Diagnosis: Submucosal lipoma, colon. (Intussusception, clinical).

COMMENT

Considering adenomatous polyps as premalignant lesions, submucosal lipomas are the most common strictly benign tumors of the colon. Yet, submucosal lipomas are relatively rare (1–28). Many surgeons will not see one in their life span.

These tumors are definite neoplasms arising from submucosal fat cells, and may be solitary or multiple. They are evenly distributed between the sexes and become clinically significant in the cancer-age group. Any part of the large bowel may be involved.

Symptomatically, they can be divided into three categories. The most important group comprises those causing obstruction. The picture of obstruction may be chronic, characterized by intermittent colic, and alternate diarrhea and constipation, depending on the degree of lumen encroachment. Acute obstruction is common and is usually based on intussusception. Melena may be present or absent. Approximately one half of the reported tumors caused obstruction.

The second group is seen in the patients who complain of bowel hemorrhage.

FIG. 1. Smooth oval defect in colon, shown by barium enema.

FIG. 2. Post-evacuation film, demonstrating intussusception of the tumor.

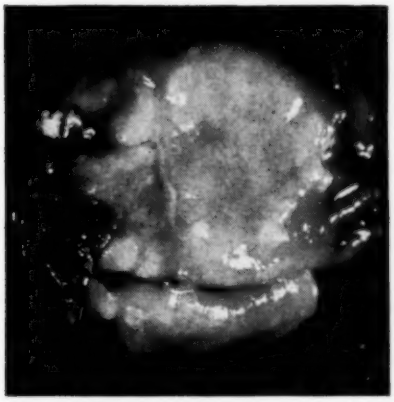

FIG. 3. Tumor removed from colon. Gross specimen, showing superficial ulceration of tip.

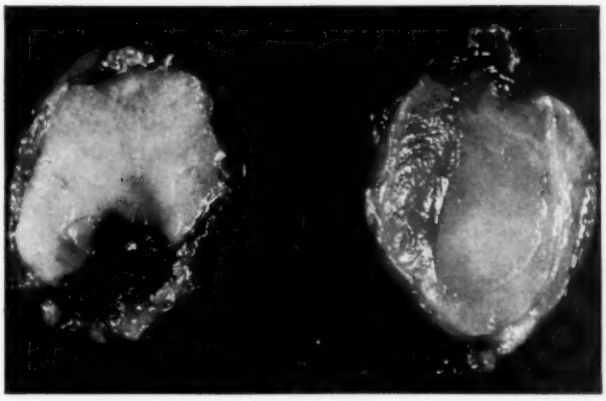

FIG. 4. Tumor—cut surface. The right half has been stained with Sudan III.

The tumors in this group are usually situated on the left side of the colon, where the firmer nature of the bowel content probably accounts for the marked ulceration.

The third group is much smaller. The patients present themselves after the tumor has undergone strangulation, infarction and expulsion.

The location of the mass, the symptomatology and the age of the patient make differentiation from malignancy impossible preoperatively. A long history of pain, change in bowel habits, melena and absence of weight loss, might make one suspicious of a benign colonic lesion. When faced with an obscure abdominal condition, the surgeon's attention should be directed to such a possibility.

Proper evaluation at the time of surgery will result in simple excision of most of these tumors, rather than segmental colectomy.

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SUPERVOLTAGE X-RAY TREATMENT OF CARCINOMA OF THE BLADDER

HUGH F. HARE, M.D.,* JOHN G. TRUMP, D.Sc.,** RICHARD C. GRANKE, B.S.** AND JOHN ANSON, M.S.†

The Los Angeles Tumor Institute, and the Albert Soiland Cancer Foundation, Los Angeles, California; the Massachusetts Institute of Technology, Cambridge, Mass.

Fortunately for those approaching the later years of life, carcinoma of the urinary bladder occurs infrequently, and there is no statistical evidence that there has been an increase in its incidence.

On the basis of symptoms alone, it is difficult to make an early diagnosis. Dysuria, frequency and hematuria are often noted, and call for a thorough investigation. When pain (other than in the bladder) is associated with this form of cancer, it is good evidence that an infiltrating lesion has spread to other locations.

Clinically, there are two classifications of malignant growths of the bladder:

1. The infiltrating
2. The non-infiltrating

Surgery has demonstrated its efficacy in the treatment of the non-infiltrating type of malignancy, controlling about 75 per cent of such cases. This is a remarkable achievement, but the unsuccessful group (25 per cent) indicates the necessity of a follow-up system and the use of other forms of therapy, so that these failures may be given adequate care.

Surgery has not controlled the infiltrating lesion—in fact, it is the opinion of many experienced surgeons that radical surgery is no longer indicated.

Histologically, these may be: 1) papilliferous, 2) squamous carcinoma, 3) adenocarcinoma, or 4) transitional-cell carcinoma.

The papilliferous lesions are the most benign group encountered; yet, if they are not properly eradicated, they become infiltrating papillary adenocarcinomas.

The adenocarcinoma, the squamous-cell carcinoma, and the transitional-cell carcinomas are all infiltrative lesions and require radical treatment, except in the early stage before definite infiltration has taken place. When they have extended deep enough to invade the muscle, the possibility of cure by local removal is remote.

The lymphatic drainage of carcinoma of the bladder is similar to that of carcinoma of the prostate and of the cervix. The lymph channels in the mucosa of the bladder may serve as avenues of spread within the organ before invasion takes place, although the possibility of multicentric origin cannot be excluded in all instances. Once the lymphatic spread has extended beyond the confines of the bladder proper, one may find involvement of the ureteral, iliac, sacral, ob-

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turator fossa, and inguinal nodes. Occasionally, one sees lymphatic spread to the vertebrae, as is common in cancer of the prostate.

Hematogenous spread occurs frequently enough to make it necessary to rule out radiographically evidence of prepneumonary or other lung lesions before treatment is instituted.

METHOD OF TREATMENT

The cases here reported were treated in cooperation with Dr. John Trump at the Massachusetts Institute of Technology. Two million volt x-ray therapy was employed. The cases were all studied by competent urologists prior to treatment, to determine the extent of involvement and the type of lesion. Most of the patients presented advanced malignant growths. In some instances, the disease was so far advanced that patients were unable to complete their treatment. Many had undergone some surgical therapy (either partial resection or electro-desiccation) but were too far advanced for radical surgery.

Surgery and electro-desiccation resulted in interference with the natural blood supply of the bladder, thus making radiation in large doses more hazardous, especially in the older age group in which arteriosclerosis is frequently a complicating factor.

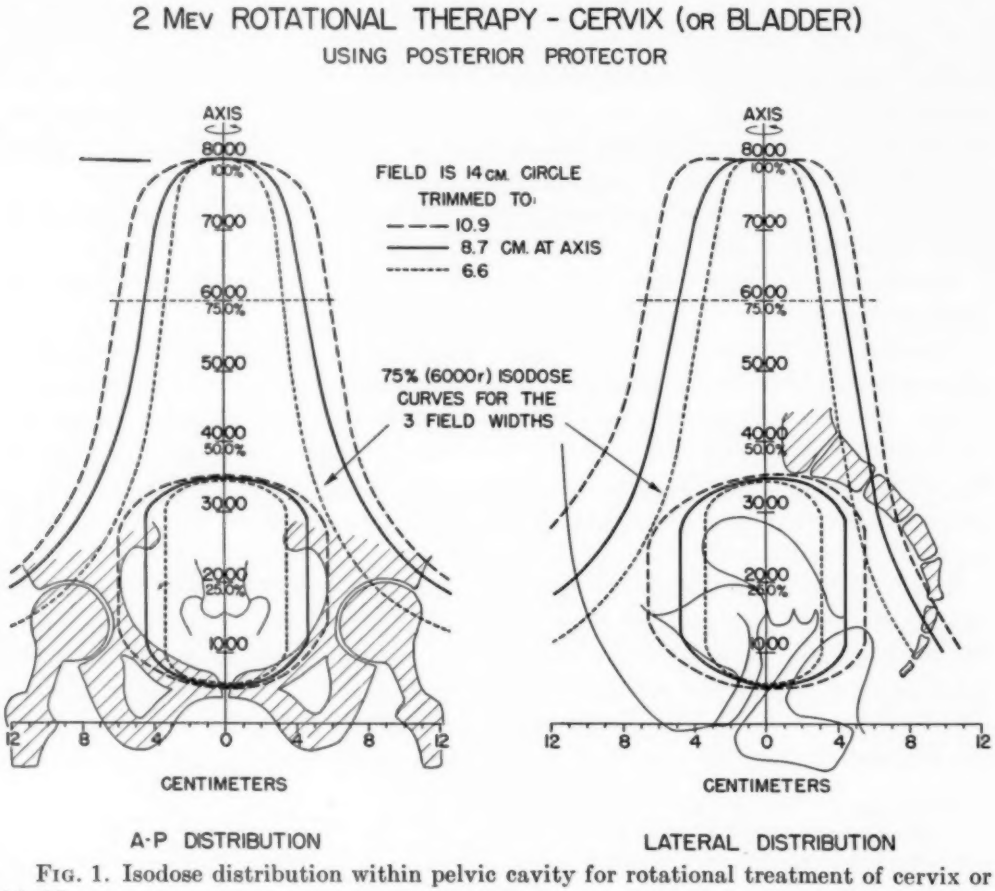

FIG. 1. Isodose distribution within pelvic cavity for rotational treatment of cervix or bladder.

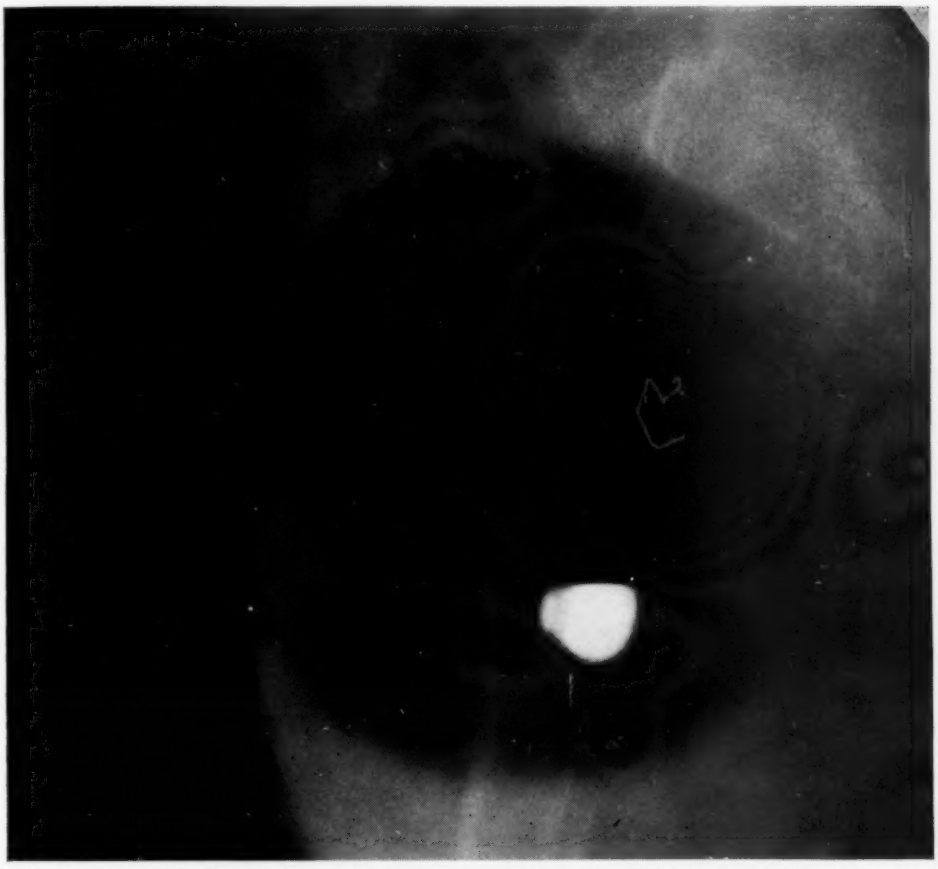

FIG. 2. Lateral radiograph of pelvic region, showing air-filled bladder and Foley catheter bulb filled with mercury for localizing treatment field.

Once the diagnosis of cancer requiring radical treatment was made, careful and complete genito-urinary studies were carried out. These included cystoscopic evaluation of the extent of the disease, rectal examination, and intravenous pyelographic studies.

In some cases, cystologic smears of the urine served as a useful aid in arriving at a diagnosis; this was also the means by which an attempt was made, in a few cases, to determine the time of disappearance of the tumor cell at various x-ray dosage levels.

In treating infiltrating cancer of the bladder rotationally with 2 million volt radiation, a total dose of 8,000 r is delivered to the bladder in thirty-five treatment days. The field is essentially cylindrical, being shaped from a 14-cm. circle by two lateral absorbers which reduce the field width to about 8 cm., depending on the size of the pelvic cavity. The dose distribution analysis in Figure 1 shows that, with such a field shape, the maximum dose is delivered to the central region and the dose diminishes to about 75 per cent of this value at the lateral pelvic wall.

In this form of rotational therapy of advanced tumors of the bladder, the patient is supported in a standing position on a rotating platform and irradiated

FIG. 3. Intravenous pyelogram demonstrating carcinoma of the bladder, with an irregular defect involving the left two-thirds; there is partial obstruction of the left ureter and hydronephrosis of the left kidney.

with a horizontal beam of appropriate shape. The vertical axis of rotation passes through the center of the primary tumor region. Rotation is at about 1 revolution per minute. Prior to treatment, the patient is placed in the Trendelenburg position and bound with a 6-inch Ace bandage, in order to minimize the amount of lower bowel in the treatment field. A posterior absorber is also used to diminish the dose sustained by the rectum; the absorber accounts for the dyssymmetry shown in the lateral view of Figure 1.

It has been found that this total dose and dose rate is generally tolerated well by the patient and is adequate for controlling most epidermoid and adenocarcinomas which are still localized within this fairly radical treatment region.

Important in this supervoltage therapy program are those procedures which insure accuracy in localizing the dose to the bladder. Foremost among these is the radiography of the patient in the treatment position with the actual 2 million volt x-ray beam. We have found that the introduction of air into the bladder,

using a Foley catheter, is an excellent means of determining the bladder position and of confirming the proper location of the axis of rotation and the shape and elevation of the field itself. Figure 2 shows a lateral view of a patient—the film being first exposed to the treatment field and then to a larger field to superimpose additional anatomic background. Such a film is taken in the first few days of treatment and is reviewed; then the positioning is modified until the treatment program has been adequately established; the film also serves as an excellent permanent record of the treatment.

The palliative results of this rotational supervoltage treatment are so encouraging that we are now advising it for all patients with infiltrative carcinoma (Fig. 3). Best results are obtained from early treatment; it also appears that the less surgical intervention prior to irradiation, the better. Of course, thorough study and biopsy are necessary.

RESULTS

The early results of treatment indicated that good palliative effects were obtained and, in nearly all cases, there was a favorable local response of the tumor, as indicated by the disappearance of pain and bleeding.

Of the 19 cases treated between October 1949 and January 1953, only 15 proved to be primary in the bladder. Seven of these patients are living and 8 are dead; among these 8, there were 3 who died of widespread metastatic disease, and 5 of the persistent bladder lesion. During follow-up observation, the recurrence or persistence became apparent from nine to forty-one months after treatment.

In our earlier articles, we considered a dose of 6,000 r to be adequate for destruction of the tumor, since negative results from cystoscopic examinations three to six months following treatment, indicated clinical disappearance of the disease. With further follow-up study—not only from the standpoint of the malignant disease, but from the standpoint of changes brought about by the radiation—the dose has been increased, but the time interval has been kept constant. It is hoped that the increased amount of radiation will bring about more lasting results and that the complications of treatment, none of which has been severe, will not be increased.

SUMMARY

Supervoltage x-ray therapy, utilizing rotation, has proved to be a useful tool in the treatment of carcinoma of the bladder. Early results indicate that it is possible to eradicate the tumor clinically, by administering a dose of 6,000 to 8,000 r. The patients receiving 6,000 r showed a good initial response, but some of them have shown a recurrence of the disease. We are, therefore, advising that the dose be increased to 8,000 r.

The complications following this treatment have not been severe, except for bladder irritation occurring in the latter part of the treatment and immediately following it.

AMERICAN GERIATRICS SOCIETY

THE 1954 ANNUAL MEETING

The 11th Annual Meeting of the American Geriatrics Society will be held at the Fairmont Hotel in San Francisco, June 17 through 19.

Hotel reservations should be made through the San Francisco Convention and Visitors Bureau, 200 Civic Auditorium, San Francisco 2, California. Members should reserve accommodations immediately, stating times of arrival and departure, because the hotels expect to be filled to capacity.

The Annual Business Meeting will be held in the San Francisco Room of the Fairmont Hotel, Thursday morning, June 17, at 9 o'clock. All scientific sessions will be held in the Terrace Room. The Annual Dinner is scheduled for the Nob Hill Room on Friday evening, June 18, at 8 o'clock, preceded by cocktails in the Cirque Room Lounge at 7 o'clock.

The program will cover many aspects of geriatric medicine. There will be several panel discussions on subjects of current interest.

Dr. Laurance W. Kinsell, Highland Alameda County Hospital, 2701 Fourteenth Avenue, Oakland 6, California, is in charge of local arrangements for the meeting.

W. O. Thompson, M.D.
President

SCIENTIFIC PROGRAM

FAIRMONT HOTEL

THURSDAY, JUNE 17, 1954

8:00 A.M. Registration (*Terrace Room Foyer*)
9:00 A.M. Annual Business Meeting (*San Francisco Room*)
2:00 P.M. General Session (*Terrace Room*)

Norris J. Heckel, M.D., *Presiding*

1. Significance and Management of Genital Prolapse in the Aged
   Goodrich C. Schauffler, M.D., Portland, Ore.
2. Uterine Bleeding After the Menopause
   C. L. Buxton, M.D., New Haven, Conn.
3. The Use of Estrogens After the Menopause
   E. Kost Shelton, M.D., Los Angeles, Calif.
4. Cancer of the Breast—A Curable Disease
   H. Glenn Bell, M.D., San Francisco, Calif.

Edward Henderson, M.D., *Presiding*

5. The Psychology of Retirement
   Edward B. Allen, M.D. and Hollis E. Clow, M.D., White Plains, N. Y.
6. Criteria for the Feasibility of Rehabilitation in the Geriatric Patient
   Edward J. Lorenze, M.D., White Plains, N. Y.

Panel Discussion

7. RECENT DEVELOPMENTS IN CARDIOLOGY
   George C. Griffith, M.D., Los Angeles, Calif., Moderator
   Francis L. Chamberlain, M.D., San Francisco, Calif.
   William Paul Thompson, M.D., Los Angeles, Calif.

FRIDAY, JUNE 18, 1954

8:00 A.M. Registration (*Terrace Room Foyer*)
9:00 A.M. General Session (*Terrace Room*)

Edward B. Allen, M.D., *Presiding*

8. The Influence of Surgery in the Aged on Surgery in the Young
   Robert Elman, M.D., St. Louis, Mo.
9. The Diagnosis and Treatment of Diseases of the Bladder in the Aged
   Norris J. Heckel, M.D., Chicago, Ill.
10. The Fate of the Cholescystostomy Patient
    Daniel M. Hays, M.D. and Frank Glenn, M.D., New York, N. Y.
11. The Medical and Surgical Management of Acute Diverticulitis in Geriatric Patients
    Robert Priest, M.D., Brock E. Brush, M.D. and Louis Gregory, M.D., Detroit, Mich.

Malford W. Thewlis, M.D., *Presiding*

12. New Trends in the Treatment of Fractures of the Hip in the Aged
    Ralph Soto-Hall, M.D., San Francisco, Calif.
13. Management of Hip Fracture in the Geriatric Patient
    Charles V. Heck, M.D., Chicago, Ill.

Panel Discussion

14. METHODS OF DETERMINING OPERABILITY IN OLDER PATIENTS
    Warren H. Cole, M.D., Chicago, Ill., Moderator
    Differences in Physiology of the Young and Aged: Their Application to Operability—*Arthur J. Hunnicutt, M.D., Oakland, Calif.*
    Cardiac and Renal Reserve in the Aged as Related to Operability—*Charles T. Stone, M.D., Galveston, Texas*
    The Use of Laboratory Tests in Determining Operability—*Dr. Cole*

INTERMISSION FOR LUNCH

2:00 P.M. General Session (*Terrace Room*)

Elizabeth Mason-Hohl, M.D., *Presiding*

15. Overtreatment in the Aged
    Malford W. Thewlis, M.D., Wakefield, R. I.

16. Clinical Experience in Restoring Verbal Communication to 266 Laryngectomized Patients by Esophageal Speech
Lynwood Heaver, M.D., Willard White and Nona Goldstein, B.A., New York, N. Y.
17. The Influence of Endocrine Substances on the Metabolism in the Older Individual
William B. Kountz, M.D. and Teofil Kheim, M.D., St. Louis, Mo.
18. A Study of the Effects of Thyroid Medication on Pathology and Longevity in Aged Human Beings
Paul Starr, M.D., Los Angeles, Calif.

Warren H. Cole, M.D., *Presiding*

19. Hormones, Growth and Senescence
Laurance W. Kinsell, M.D., Oakland, Calif.

Panel Discussion

20. CHANGES IN STEROID EXCRETION WITH ADVANCING YEARS
Cornelius P. Rhoads, M.D., New York, N. Y., Moderator
Technical Advances in Steroid Measurement—*Thomas F. Gallagher, Ph.D., New York, N. Y.*
The Chromatograms of Urinary 17-Ketosteroids in Aged Persons Before and After the Administration of Stanolone and Testosterone —*Thomas H. McGavack, M.D., Sidney Pearson, Ph.D. and Helmut Haar, M.D., New York, N. Y.*
Adrenocortical Capacity and Metabolism of Hydrocortisone in Elderly Patients—*Frank Tyler, M.D., Salt Lake City, Utah*

ANNUAL DINNER, Friday, June 18, 1954

7:00 P.M. Cocktails—Cirque Room Lounge—Sponsored by the Schering Corporation
(Attendance limited to holders of dinner tickets)

8:00 P.M. Dinner (Dress optional)— Nob Hill Room
The dinner wine will be supplied by Wente Wineries, and the dessert wine by Cresta Blanca Wineries.

SATURDAY, JUNE 19, 1954

8:00 A.M. Registration (*Terrace Room Foyer*)
9:00 A.M. General Session (*Terrace Room*)

Hollis E. Clow, M.D., *Presiding*

21. Management of the Burned Elderly Patient
Sidney E. Ziffren, M.D., Iowa City, Iowa
22. Histologic Patterns of the Gastrointestinal Tract in Various Age Groups
Edward Henderson, M.D., Bloomfield, N. J.
23. A New Therapeutic Approach to the Management of Debilitating Diseases
Cortez F. Enloe, Jr., M.D., New York, N. Y.

Francis M. Pottenger, Jr., M.D., *Presiding*

Panel Discussion

24. THE RELATION OF INDUSTRY TO GERIATRICS
Carl M. Peterson, M.D., Chicago, Ill., Moderator

WHO SHOULD RETIRE?—*Charles E. Dutchess, M.D., New York, N. Y.*

THE IMPORTANCE OF GERIATRICS IN INDUSTRIAL MEDICINE—*Rutherford T. Johnstone, M.D., Los Angeles, Calif.*

THE STRESS DISORDERS—*William P. Shepard, M.D., New York, N. Y.*

25. TEMPORAL ARTERITIS
Bayard T. Horton, M.D. and Thomas B. Magath, M.D., Rochester, Minn.
26. PROGNOSIS AND TREATMENT OF OCULAR DEFECTS OF THE AGED
A. E. Maumenee, M.D., San Francisco, Calif.

TO BE READ BY TITLE

27. TUBE FEEDING WITH LIQUEFIED WHOLE FOOD IN ELDERLY PATIENTS
J. Barron, M.D. and L. S. Fallis, M.D., Detroit, Mich.
28. USE OF PSYCHOLOGIC TESTS IN DETERMINING PROGNOSIS AND TREATMENT IN GERIATRIC MENTAL ILLNESSES
D. V. Conwell, M.D., C. J. Kurth, M.D. and P. G. Murphy, Ph.D., Wichita, Kans.
29. MARRIAGE COUNSELLING IN GERIATRICS
A. K. Daniels, M.D., San Francisco, Calif.
30. STUDIES OF PLASMA HEPARIN LEVELS IN RELATION TO SEX, AGE, AND SERUM LIPIDS AND LIPOPROTEINS
H. Engelberg, M.D., Los Angeles, Calif.
31. BACTERIAL ALLERGY AS A CAUSE OF SO-CALLED INTRINSIC ASTHMA IN THE ELDERLY PATIENT
J. Forman, M.D. and H. Blatt, M.D., Columbus, Ohio
32. ARTERIAL DISEASE IN THE AGED
R. S. Gilfillan, M.D., San Francisco, Calif.
33. X-RAY FINDINGS IN GERIATRICS SURVEYS
G. J. Heppner, M.D., San Francisco, Calif.
34. STUDY OF THE PROBLEM OF PROSTHETIC FITTING AND TRAINING OF THE GERIATRIC PATIENT
M. K. Newman, M.D., Detroit, Mich.
35. SCREENING IN GERIATRICS
R. M. Perlman, M.D., San Francisco, Calif.
36. GERIATRIST-PHYSIATRIST TEAM IN GERIATRICS REHABILITATION
R. M. Perlman, M.D. and S. M. Dorinson, M.D., San Francisco, Calif.
37. BALLISTOCARDIOGRAPHY IN GERIATRICS
B. Schneider, M.D., Danville, Pa.
38. FURTHER CLINICAL STUDIES OF THE EFFECTS OF METRAZOL AND NICOTINAMIDE ON PSYCHIC AND MENTAL DISORDERS OF THE GERIATRIC PATIENT
Herman Seidel, M.D., Abraham A. Silver, M.D., Henry Nagel, M.D. and Jerome J. Blumberg, M.D., Baltimore, Md.

SCIENTIFIC SESSIONS ARE OPEN TO THE MEDICAL PROFESSION AND TO THOSE WHO ARE ESPECIALLY INTERESTED IN THE PROBLEMS OF AGING

THIRD INTERNATIONAL GERONTOLOGICAL CONGRESS—LONDON

The Third International Gerontological Congress will be held in London, England, *July 19–23, 1954*, under the presidency of Dr. J. H. Sheldon. Arrangements by the British Organizing Committee are well advanced. Dr. R. E. Tunbridge, B.M.A. House, Tavistock Square, London W.C.1, is Chairman of the Committee. An *American Committee* is cooperating with the British Committee, especially in regard to the program. Of this Committee *Dr. W. B. Kountz, 660 S. Kingshighway Blvd., St. Louis 10, Mo., is the Chairman.* The Congress will be both instructive and enjoyable. It is imperative for those planning to attend and to present their work, to send titles of papers, together with abstracts of not more than 250 words, to Dr. Kountz. As in the case of other International Congresses, personal invitations to present papers are not necessary but in the event that these would be helpful in obtaining subsidies for travel, the American Committee will be happy to request the British Organizing Committee to send official invitations.

The Research Committee on Biology and Medicine will meet at the same time as the Third International Gerontological Congress. The Chairman of this Committee is Dr. W. O. Thompson, Chicago, Illinois.

Because the time is short and it is difficult to get transatlantic reservations, except the more expensive ones, blocks of reservations are being held by Convoys Travel Service, 1133 Broadway, New York 10, N. Y. All correspondence concerning *travel information* should be addressed to *Mrs. V. Kelly, 660 S. Kingshighway, St. Louis 10, Mo.*

PROVISIONAL PROGRAM

July 19, A.M. Meeting of the Governing Body.
July 19, P.M. Official opening of Conference, followed by meetings of the Sectional Committees.
July 20, 21 and 22. Scientific Meetings of the Conference.
July 23. Closing Meetings of the Conference.

The meetings and discussions will be divided between three main sections of the Conference:

Section 1. Biology and Pathology.
Section 2. Geriatric Medicine and Psychiatry.
Section 3. Sociology and Psychology.

Discussions and symposia involving combined meetings of two or more sections, and meetings of the Research Group, are to be arranged.

Visits

Visits will be arranged during the meeting in London, to centers in and around London. After the Congress, tours will be arranged to other centers in the United Kingdom.

Opportunities will be given to members to see geriatric units in hospitals, residential homes, housing accommodations, clubs and special industrial facilities.

Proposed Provincial Tours

Following the main Congress, tours will be arranged to enable members to see something of the work for the elderly throughout the country. Local members have expressed the wish to meet members attending the Congress and to entertain them during the provincial visits.

The following tours are being arranged: 1). London, Oxford, then to the West Country, returning via Southampton and Salisbury to London. 2). London, Nottingham, Sheffield, Leeds, Harrogate, Durham, Sunderland, York, Lincoln and Cambridge, to London.

Social Occasions

July 19. President's Reception in the evening.
July 21. Evening Reception.
July 23. Conference Dinner.

Throughout the Congress, visits to centers of scenic and historic interest in and around London will be arranged. Opportunities will also be provided for members to meet informally.

RESEARCH COMMITTEE ON BIOLOGY AND MEDICINE
OF THE
INTERNATIONAL ASSOCIATION OF GERONTOLOGY

REPORT OF MEETING OF THE AMERICAN DIVISION, SAN FRANCISCO, AUGUST 25, 1953

A short meeting of the American section of the Research Committee on Biology and Medicine of the International Association of Gerontology was held during the meeting of the Gerontological Society, Incorporated, in San Francisco at the Marks Hopkins Hotel, Tuesday, August 25, 1953. The following members were present:

Dr. Warren Andrew
Dr. E. V. Cowdry
Dr. John Esben Kirk
Dr. Albert I. Lansing
Dr. N. W. Shock
Dr. W. O. Thompson, Chairman

These members of the Committee expressed interest in the ideas already set forth by Dr. V. Korenchevsky concerning the objectives of the Research Committee. Further consideration of the problem at the time of the meeting of the International Congress in London, England, July 19–23, 1954, was considered desirable.

It was felt that elaborate plans for the meeting in London could not be made at the present time because no funds are available for traveling expenses of members of the Committee. In most instances, American investigators and clinicians who wish to attend the meeting in London must do so at their own expense.

W. O. Thompson, M.D.
Chairman, Research Committee on Biology and Medicine

MEDICAL LIBRARY ASSOCIATION

The Medical Library Association will hold its Fifty-third Annual Meeting June 15–18, 1954, in Washington, D. C. The headquarters will be the Hotel Statler, and the official host the Armed Forces Medical Library.

The program will include a discussion on medical research by embassy attaches, tours of the National Institutes of Health, the National Naval Medical Center, and the Armed Forces Medical Library. Delegates to the Meeting will hear addresses by Dr. Detlev Bronk, President of the Rockefeller Institute of Medical Research, Lt. Col. Frank B. Rogers, Director of the Armed Forces Medical Library, Mr. Verner Clapp, the Acting Librarian of Congress, and Dr. Raymond Zwemer, Chief of the Library of Congress' Science Division.

Further information can be obtained from Lt. Col. Frank B. Rogers, Armed Forces Medical Library, 7th Street and Independence Avenue, S. W., Washington 25, D. C.